

NATIONAL RESEARCH COUNCIL  
Inadequate Diets and Nutritional Deficiencies  
in the United States

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INADEQUATE DIETS AND NUTRITIONAL  
DEFICIENCIES IN THE UNITED STATES

THEIR PREVALENCE AND SIGNIFICANCE

REPORT OF THE COMMITTEE ON DIAGNOSIS AND  
PATHOLOGY OF NUTRITIONAL DEFICIENCIES  
FOOD AND NUTRITION BOARD

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## PREFACE

During the past decade the results of dietary surveys and nutritional appraisal among the population have been reported at irregular intervals in widely scattered journals; hence their full weight in the aggregate has scarcely been considered. For convenient reference but more for provision of proper perspective, it was thought useful to bring together all the available results.

In addition, this review contains a considerable amount of previously unpublished material. Because of lack of uniformity in selection of standards and in presentation, the results of most diet studies as originally recorded could not be compared. For this review several authors at much effort recalculated and reclassified their data on a uniform, authoritative and comparable basis. Here the results appear in this form for the first time. To these authors the Committee on Diagnosis and Pathology of Nutritional Deficiencies expresses its deepest appreciation.

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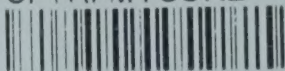
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## THE PREVALENCE OF INADEQUATE DIETS AS SHOWN BY SURVEYS

Every nutritional survey in the past decade has revealed that the consumption of diets below the recommended standards is widespread in the United States. In 1933 in New York City, 48 percent of the low-income families canvassed had an average daily caloric intake below that estimated to be the minimum requirement; <sup>1, 2</sup> 26 percent had diets yielding an energy value by approximately 20 percent or more under that level. In the same year a survey of low-income families in Baltimore, Cleveland, Detroit, Pittsburgh, and Syracuse showed that 27 percent of them had an average daily caloric intake lower than the minimum standard; <sup>2, 3</sup> 18 percent had diets with a caloric value approximately 20 percent or more below that level. Two-thirds of all the families surveyed consumed diets at or below the minimum standard level for several protective foods. It is to be noted that the minimum level, whether for energy or protective foods, allowed no margin of safety. Consequently, the proportion of families with unsatisfactory diets was certainly much higher than has been represented.

Among families of employed wage-earners and clerical workers in 43 industrial centers in eight major geographical regions, it was found during 1935 and 1936 that the maximum proportion of white families with good diets in any region was only 21 percent.<sup>4</sup> In the region with the poorest showing, no more than 11 percent of the families had good diets. Between these two, the percentages in other regions were intermediate. Only 11 percent of the colored families had good diets.

In the Committee's interpretation of the rating of diets and the standards upon which it is based (Table 1), anything less than good was considered as not satisfactory; \* consequently, the percentage of families with unsatisfactory diets was extremely high. Indeed, by the original rating the proportion of families having diets rated as poor † ranged from 40 percent of the white families in one region, as the least, to 64 percent of the colored families in another region, as the greatest.

\* According to the system of grading (Table 1) specified in the original paper,<sup>4</sup> fair diets more than met minimum requirements, but were inferior to good diets. Here fair is used in a relative sense. In their interpretation of such a relative scale, persons are apt to attribute a value to a diet which it does not possess on an absolute basis. To many persons fair may appear to have a certain amount of goodness and to be regarded as satisfactory. Rather it expresses here a degree of inferiority. Especially when a diet falls below a critical level, its relative value of fair is less important than that on an absolute basis it is unsatisfactory. It is doubtful that minimum requirements for any extended period would confer protection against deficiency states. Consequently, meeting basic requirements which are inadequate for any prolonged period is no recommendation of goodness. A diet that falls short, even so slightly, and permits development of deficiency states, may be relatively fair but absolutely it is unsafe. According to the standards of the study, the Committee regards fair diets as unsatisfactory.

† Since these diets failed to meet minimum requirements, a level that will not protect for any protracted period, the Committee would regard them as very poor diets.



riboflavin, and 30 percent with respect to vitamin A. Even on farms, one-fourth fell short of the recommended dietary goals in calcium and/or vitamin A, and one-half or more with respect to thiamin, riboflavin, and/or ascorbic acid. Because neither food wastes nor nutritive loss in preparation was taken into account, it was acknowledged that all the figures on dietary adequacy as well as those showing the proportion of families meeting the Council's recommendations tend to be optimistic.

Among 538 farm and 299 village families of Vermont visited in 1936, inadequacy in consumption of fruits and vegetables was found to be common; while low intake of protein occurred in families with children.<sup>11</sup>

Of 100 submetropolitan urban families in Central and Western Pennsylvania surveyed in 1935-37, it was found<sup>12</sup> that the following percentages had diets containing the several requisite nutrients in amounts less than those recommended by the National Research Council: riboflavin, 59; calcium, 58; calories, 49; thiamin, 37; vitamin A, 33; protein, 29; iron, 21; and ascorbic acid, 7.

Inquiry into the diets of pupils in Wilkes Barre, Pennsylvania, in 1937 showed<sup>13</sup> that the following percentages of 748 white children, 1 to 12 years old, were receiving amounts of essentials below the Council's suggested levels: riboflavin, 99; calories, 93; calcium, 92; protein, 81; iron, 81; thiamin, 74; ascorbic acid, 71; and vitamin A, 67. Of 39 white adolescents, 13 to 20 years old, the percentages were: calories, 100; protein, 100; calcium, 100; thiamin, 100; riboflavin, 100; iron, 97; ascorbic acid, 95; and vitamin A, 85. Corresponding percentages for 14 colored children, 1 to 12 years old, were: calories, 100; calcium, 100; riboflavin, 100; protein, 92; iron, 92; thiamin, 85; ascorbic acid, 85; and vitamin A, 54.

#### DIET SURVEYS CONDUCTED IN YEARS 1938-40

In a survey in 1938 of families on relief in Washington, D. C., it was found that the average daily intake of 87 percent of the white families was less than 2,800 calories per consumption unit, a standard little if any in excess of actual needs.<sup>14</sup> The diets of both white and colored families were extremely low in calcium and only slightly better in iron. Diets as low as these in energy value certainly were deficient in most nutrients.

Diet records of elementary school children in a Maine village—76 in autumn, 1938 and 63 in spring, 1939—showed that only one child in seven attained the minimum standard of one good vitamin C food daily.<sup>15</sup> In the autumn only 17 of the 76 children ate any citrus fruit, the best source of vitamin C, during the week of the record. In the spring, half of the children had some citrus fruit; but only 14 of the 63 had it oftener than once or twice in the week.

To obtain adequate amounts of various essential nutrients, certain types of foods must be eaten in sufficient quantities. Although most of the essential nutrients are distributed in nature in many different foods, some are present in appreciable amounts in only a few types of foods, others only in one type of food. Thus, some essentials to be obtained in sufficient quantities must



come from two or three types of food, none of which eaten alone in usual quantities supplies the necessary amount. Other essentials are obtained principally from one type of food. Conversely, a type of food may be the almost exclusive source of a necessary nutrient, or the indispensable source of much of an essential nutrient, or the exclusive source of one and the supplementary source of another nutrient. The several types of food necessary to provide all nutrients form the foundation of the dietary structure. They are essential types of foods because each is the main or indispensable source of one or more requisites.

Unless citrus fruits or tomatoes are included in the diet, it is almost impossible to meet the Council's recommended amounts of vitamin C. Enriched or whole-grain bread or a whole-grain cereal is needed to ensure an adequate supply of vitamin B<sub>1</sub>. A green or yellow vegetable furnishes a substantial proportion of vitamin A; it also contains some vitamin B<sub>1</sub>. An adequate quantity of milk provides most of the calcium; it supplies a substantial proportion of riboflavin; it also furnishes a considerable amount of vitamin A supplementing that from vegetables; and it yields some vitamin B<sub>1</sub>. If milk were the sole source of vitamin A in a diet, it would have to be consumed in excessively large quantities to supply the necessary amount of that nutrient. Meat is the main source of niacin; it also provides a considerable supplement of riboflavin; and supplies some vitamin B<sub>1</sub>. If it were the sole source of riboflavin in a diet lacking milk, meat would have to be eaten in exceptionally large amounts. A regular and adequate supply of the principal vitamins and minerals is most readily obtained by consumption of these foods in sufficient amount each day. Otherwise, the diet must be specially planned to furnish all the necessary nutrients.

In its report on Recommended Dietary Allowances<sup>7</sup> the National Research Council presented two sample dietary patterns to show the varied ways in which allowances may be met (Table 2). List I contains the commonly available types of food. Other combinations of food expertly planned, as in List II, will also cover the allowances.

Of 3,432 women college students in six institutions in the North Central States<sup>16</sup> canvassed during 1936 to 1940, the following percentages had diets containing less than seven servings a week of the respective essential types of foods: whole grain products, 66; citrus fruits, 57; green and yellow vegetables, 40; milk, 29; and meat, 14.

Among 19 rural families in Lancaster County, Pennsylvania, in 1938-1940, it was found<sup>17</sup> that the following percentages of 42 white children, 1 to 12 years old, were eating less than the amounts of the several respective constituents advised by the Board: riboflavin, 81; calcium, 64; calories, 36; thiamin, 36; vitamin A, 33; ascorbic acid, 24; protein, 21; and iron, 17. For 20 white adolescents, 13 to 20 years old, the corresponding percentages were: riboflavin, 90; calcium, 70; vitamin A, 45; thiamin, 45; calories, 30; ascorbic acid, 30; protein, 20; and iron, 20. For 38 white adults the percentages were: riboflavin, 97; calories, 87; thiamin, 84; vitamin A, 37; calcium, 24; iron, 10; protein, 8; and ascorbic acid, 5.



In a survey during 1939-40 of over 2,000 high school pupils of low-income families in New York City, 74 percent of the pupils had a daily caloric intake below their individually calculated energy needs; 21 percent had less than two-thirds of their computed caloric requirements;<sup>18</sup> 44 percent, 64, and 72 percent had less than the Council's recommended daily allowances of protein, iron, and calcium, respectively; 5, 17 (29 percent of the girls), and 28 percent had less than two-thirds of the approved amounts of these respective nutrients.

TABLE 2

SAMPLE DIETARY PATTERNS, PREPARED BY FOOD AND NUTRITION BOARD, NATIONAL RESEARCH COUNCIL,<sup>7</sup> TO SHOW WAYS IN WHICH ALLOWANCES MAY BE MET

## LIST I

Milk .....	1 pint
Egg .....	1 daily, if possible. (On days not used, beans, peanuts, cheese, or more milk or meat to be used instead)
Meat, fish or fowl.....	1 or more servings
Potato .....	1 or more
Vegetables .....	2 or more servings. One green or yellow
Fruits .....	2 or more. One citrus fruit or tomato or other good source of vitamin C
Cereals and bread.....	Whole-grain or enriched
Other foods as needed to complete the meals.	

This list is based on the needs of the average adult.

## LIST II

Turnip greens .....	1 cup
Sweet potatoes .....	3
Peanuts .....	20 nuts or 2 tablespoons of peanut butter
Beans or cowpeas.....	1½ oz.
Tomatoes .....	1 cup
Corn meal .....	3 oz.
Enriched flour .....	3 to 4 oz.
Milk (fresh, evaporated or dried)....	⅓ qt.
Lean pork .....	Small serving 3 to 4 times a week
Molasses, fat, etc., to complete the meals.	

The percentages of pupils having less than the standard daily amounts of the several vitamins were: 65 for vitamin A, 53 for vitamin B<sub>1</sub>, 70 for riboflavin, and 58 for vitamin C. The percentages with a daily intake of less than two-thirds of the recommended amounts of these essentials were: 38 for vitamin A, 14 for vitamin B<sub>1</sub>, 25 for riboflavin, and 29 for vitamin C. It should be noted that a daily intake of essential nutrients less than two-thirds of the advocated allowances probably does not meet even minimum requirements which are not regarded as safe levels.

In contrast,<sup>19</sup> among 293 private school pupils the following percentages had less than the recommended levels of the several essentials: calories, 68; calcium, 61; iron, 36; riboflavin, 36; vitamin A, 35; protein, 26; ascorbic acid, 20; and thiamin, 18, respectively. It will be noted that for every essential the percentage of pupils receiving a substandard amount was less, in most



instances much less, in this group from high-income families than in the preceding group from low-income families. But even among the group from high-income families, the percentage of pupils with diets that failed to meet accepted standards of adequacy was appreciable.

A survey of dietary records of 1,082 Farm Security Administration families in Louisiana <sup>20</sup> during 1939 and 1940 showed less than 1 percent which could be classified as good, while 52 percent were fair and 47 percent were poor. Of 573 white families, 1 percent had diets classed as good; 69 percent as fair; 30 percent as poor. Among 509 colored families, none had good diets; 32 percent, fair; and 68 percent, poor. By using a modification of the pattern established by the National Research Council, a score card was developed for evaluating the adequacy of the diets. A diet was rated good, fair, or poor, depending on the number of times each of the types of essential foods was served during the week. A poor diet was distinctly deficient in one or more of the food factors necessary for optimum health. It was stated that diets classed as fair might be defined as borderline diets. An active individual eating such a diet would not receive enough of the various essential substances to meet adequately his requirement. It is notable that citrus fruits and tomatoes were served least frequently.

Among 225 private patients in the upper-income levels,<sup>21</sup> surveyed in Philadelphia in 1940, the following percentages had diets containing less than the Board's recommended allowances for respective nutrients: riboflavin, 77; thiamin, 76; calories, 74; calcium, 46; iron, 40; protein, 37; vitamin A, 26; ascorbic acid, 13. Seven percent of the patients consumed less than the Board's prescribed amounts of all eight factors. The following percentages of the patients had less than 50 percent of the Council's recommended levels of the various constituents: riboflavin, 26; thiamin, 13; calcium, 13; calories, 5; vitamin A, 5; ascorbic acid, 4. In the surveyed group were nine physicians whose diets were as bad as those of the other patients.

#### EVIDENCE FROM DIETARY SURVEYS DURING PAST THREE YEARS

From January, 1939 to August, 1941 the dietary habits of 7,363 children and adolescents from 3,922 families in Chicago were surveyed.<sup>22</sup> They represented different national and racial groups and a wide distribution of economic and social conditions in the city. Seventy-six percent were white; 19 percent, Negro; 5 percent, Mexican.

The diet patterns of 72 percent of the children failed to meet a standard lower than that recommended by the National Research Council. Ninety-three percent of the Mexican children, 89 percent of the Negro, and 67 percent of the white were eating diets inferior to even the low standard.

Dietary inadequacies were least often noted in the protein foods and most often in fruits and vegetables. Regardless of which of several criteria was adopted for determining adequacy, a large proportion of the diets fell below the standard in the latter foods. The percentages of children with diets that included less than the low requirements of the different food groups were: for meat, fish, and eggs, 15; for milk, 37; for fruits and vegetables, including



potatoes, 65. Compared with the National Research Council's recommendations, the diets of 73 percent of the white children were below the allowances for fruits and vegetables, and in the poorest areas 98 percent were below this standard (Table 3).

Analysis of records on 110 rural adults in Chatham County, North Carolina, surveyed in 1940-41, revealed a high prevalence of unsatisfactory diets.<sup>23, 24</sup> Almost all persons received less than the recommended daily amounts of the several nutrients: thiamin, 99; riboflavin, 97; vitamin C and calcium, 84; protein, 83; iron, 82; and vitamin A, 70 percent, respectively. Many ate less than half the recommended dietary allowances of the various essentials, as shown by the following percentages; vitamin C, 72; riboflavin, 71; calcium, 34; vitamin A, 27; protein, 10; and iron, 8. A considerable proportion of the persons obtained less than one-quarter of the standard daily amounts of the nutrients: vitamin C, 45; riboflavin, 23; thiamin, 17; vitamin A, 9; calcium, 4; and protein, 1 percent of the subjects, respectively.

TABLE 3

PERCENTAGE OF WHITE CHILDREN FROM RELIEF FAMILIES AND NON-RELIEF FAMILIES IN DIFFERENT TYPES OF COMMUNITY AREAS IN CHICAGO, WHO CONSUMED AMOUNTS OF PROTECTIVE FOODS BELOW THE NATIONAL RESEARCH COUNCIL'S RECOMMENDED ALLOWANCES

	Relief (382)	Non-relief				
		Poor area (122)	Fair area (465)	Fair- good area (381)	Good area (277)	Very good area (565)
Fruits, vegetables .....	99.7	98.4	78.1	68.0	56.0	39.5
Milk .....	40.1	35.2	19.8	13.6	12.3	8.0
Meat, fish, poultry, eggs.....	42.0	14.8	12.5	11.5	10.1	9.5

From a study of families, both white and colored, in a rural community in middle Tennessee in 1941, diet records were obtained on 1,161 persons.<sup>25-27</sup> Of 206 white infants and children from ages 1 to 12 years, the following percentages were receiving less than the recommended daily amounts of the several nutrients: thiamin, 79; calcium, 77; iron, 70; calories, 69; ascorbic acid, 67; protein, 61; riboflavin, 55; and vitamin A, 45. For 115 colored infants and children in the same age group, the percentages receiving daily amounts of the essentials under the approved level were: calcium, 99; calories, 95; thiamin, 93; riboflavin, 91; protein, 87; iron, 83; ascorbic acid, 70; and vitamin A, 51. The percentages of white infants and children eating less than half the advised daily allowances of the various essentials were: calcium, 30; ascorbic acid, 28; thiamin, 23; riboflavin, 21; vitamin A, 18; calories, 12; protein, 12; and iron, 9. Much greater were the percentages of colored infants and children receiving less than half the recommended daily levels, as shown by the following figures: calcium, 70; riboflavin, 52; thiamin, 47; calories, 45; protein, 35; ascorbic acid, 34; vitamin A, 34, and iron, 24.

Of 113 white adolescents from ages 13 to 20, the following percentages were not receiving the suggested daily amounts of the various nutrients: thiamin, 93; calories, 89; calcium, 87; riboflavin, 85; iron, 84; protein, 78; ascorbic acid, 68; and vitamin A, 60. The following proportions of 70 colored adoles-



cents in the same age group were failing to obtain the advocated daily levels of the several constituents: calories, 100; riboflavin, 97; thiamin, 96; protein, 94; calcium, 93; iron, 89; ascorbic acid, 73; and vitamin A, 59 percent, respectively. Receiving less than half the recommended daily allowances of the various essentials were the following percentages of white adolescents: calcium, 43; riboflavin, 41; thiamin, 29; ascorbic acid, 29; vitamin A, 19; iron, 16; protein, 13; and calories, 11. The percentages of colored adolescents eating less than half the standard daily allowances of the respective nutrients were: riboflavin, 62; thiamin, 55; calcium, 45; ascorbic acid, 42; calories, 42; vitamin A, 37; protein, 33; and iron, 32.

Of 457 white adults 21 or more years old, the following percentages were not eating the recommended daily amounts of the respective constituents: thiamin, 94; riboflavin, 91; calories, 82; calcium, 61; iron, 59; ascorbic acid, 52; protein, 51; and vitamin A, 45. For 194 colored adults, the percentages receiving less than the advised daily levels of the nutrients were: riboflavin, 99; thiamin, 98; calories, 95; calcium, 77; protein, 74; iron, 69; ascorbic

TABLE 4

PERCENTAGES OF CHILDREN OF NEWPORT, MAINE, AND OF FRESHMAN GIRLS OF UNIVERSITY OF MAINE EATING LESS THAN RECOMMENDED AMOUNTS OF ESSENTIAL FOODS

Subjects	Leafy, green, and yellow vegetables	Citrus fruits and tomatoes	Other fruits and vegetables	Milk	Whole grain cereal and bread	Meat, fish, and poultry
Newport children .....	93	80	71	71	62	35
College freshman girls.....	44	19	22	39	69	0

acid, 62; and vitamin A, 48. Not a few of the white adults were eating less than half the approved daily amounts of the various nutrients, as shown by the following percentages of the persons: riboflavin, 50; thiamin, 31; calcium, 23; ascorbic acid, 21; vitamin A, 15; calories, 12; protein, 7; and iron, 7. Even greater percentages of colored adults were receiving less than half the recommended daily levels: riboflavin, 77; thiamin, 65; calories, 40; calcium, 40; ascorbic acid, 35; vitamin A, 32; protein, 18; and iron, 17.

Among children of Newport, Maine, in 1940 and 59 University of Maine freshman girls in 1941, the percentages eating the respective essential kinds of foods in amounts below the standards for a good diet <sup>28, 29</sup> are shown in Table 4.

A dietary survey of 1,169 school children in Worcester County, Maryland,<sup>30</sup> in the winter of 1941 revealed that the following percentages were not receiving satisfactory amounts of the various types of food: vegetables other than potato, 81; fruit, 80; cereal or whole grain, 59; two or more cups of milk a day, 59. In contrast, only 3 percent of the children did not obtain sufficient meat.

Only 8 percent of the children had a diet adequate in all five factors studied. Twenty-three percent were totally deficient in one factor; 35 percent in two; 27 percent in three; and 8 percent in four.

In a study of 780 families of Home Demonstration Club members in twenty-seven parishes of Louisiana <sup>31</sup> in March, 1942, 41 percent had diets scored



good; 51 percent, fair; 8 percent, poor. The diets were evaluated by comparing the frequency with which the several types of protective foods were served with the schedule formulated by the National Research Council. In such scoring, it was stated, a poor diet was distinctly deficient in one or more of the food factors necessary for optimum health; while a fair diet did not provide enough of the various food factors to meet adequately the requirements. The percentages of families eating less than the recommended amounts of each of the various types of essential foods are shown in Table 5.

The eating habits of 6,708 grade and high school students—5,255 white, 1,453 colored—in Louisiana<sup>32</sup> in 1942 were evaluated as in the preceding study by the dietary pattern recommended by the National Research Council.

TABLE 5

PERCENTAGES OF FAMILIES OF HOME DEMONSTRATION CLUB MEMBERS AND GRADE SCHOOL AND HIGH SCHOOL STUDENTS IN LOUISIANA, EATING LESS THAN RECOMMENDED AMOUNTS OF ESSENTIAL FOODS

Types of essential foods	Home demonstration club members' families (31)	Grade school and high school students (32)	
		White	Colored
Whole grain cereals.....	86	92	95
Potatoes .....	54	84	92
Butter .....	53	66	95
Citrus fruits and tomatoes.....	50	75	91
Other fruits .....	45	68	83
Other vegetables .....	41	76	86
Green or yellow vegetables.....	34	51	79
Milk .....	29	56	91
Eggs .....	28	77	84
Meat .....	20	16	31

Only 11 percent of the white and 3 percent of the Negro children received a diet adequate in all essential food factors. The diets were fair (borderline) among 60 percent of the white and 35 percent of the Negro students; poor, definitely deficient, in one or more essential nutrients among 29 percent of the white and 62 percent of the Negro children. Diets scored as fair or poor were not regarded as satisfactory. In Table 5 are shown the percentages of students eating less than the recommended amounts of each of the various types of essential foods.

From a survey of 315 colored families in the Harlem district of New York City<sup>33</sup> in 1942 were reported the following percentages on ratings for food habits: excellent, 8; good, 19; fair, 34; and poor, 39. The four ratings were based on the extent to which their food met in kind, amount, and frequency the Council's recommendations. Here again the conventional designations given to the four ratings express relative gradations: hence, the rating of fair, and possibly good, should not be construed as satisfactory. Accordingly, at least 73 percent of the families had unsatisfactory diets.

In 1941-42, the diets of 1,080 workers in the Lockheed Aircraft Corporation at Burbank, California, were analyzed for their content of both protective foods and essential nutrients.<sup>34</sup> Unless expertly planned for special purposes



the diet should regularly contain certain types of foods in adequate amounts; otherwise it is almost certain not to provide recommended amounts of the various essentials. These criteria were applied to the data from the dietary survey of the Lockheed Aircraft workers. Only 36 percent of the men had seven or more servings of citrus fruits or tomatoes during a week. It is surprising that 23 percent had no citrus fruit during a week. About 20 percent had four or more slices a day of whole wheat, rye, or dark bread. Only 7 percent ate a whole grain cereal every day; slightly over 50 percent used no whole grain breakfast cereal in a week. Only 11 percent of the men ate regularly vegetables containing vitamin A. Twenty-four percent had them less than three times a week. Sixty-three percent reported an average daily consumption of milk less than the accepted standard; 37 percent had an average of less than one-half the recommended amount; 11 percent did not drink milk. Eighty percent of the men ate meat with recommended frequency; an additional 15 percent had nearly the recommended frequency.

The frequency of diets equal to or slightly below the recommended amounts for these types of food and therefore receiving a satisfactory rating for them was as follows: vegetables, 21; citrus fruits or tomatoes, 36; milk, 51; and lean meat, 95 percent, respectively. Unsatisfactory ratings for amounts of specific foods were given to the following percentages of the diets: vegetables, 56; citrus fruits or tomatoes, 49; milk, 33; and meat, 1.

It is evident that a large proportion of the diets were low in their content of citrus fruits or tomatoes, vegetables, and milk; hence, they would be unsatisfactory in their content of vitamin A, ascorbic acid, riboflavin and calcium. Indeed, among the foods eaten in less than recommended amounts vegetables and citrus fruits were outstanding. Only 2 percent of the men had diets which approximated the recommended dietary pattern. While 11 percent of the men reported marginal diets, 87 percent had diets which were unsatisfactory for one or more food types.

One-fourth to one-third of the men probably had insufficient calories in the two-day period for which the quantitative diet was reported.

For the various nutrients, there was a great difference in the percentage of diets which met the recommended allowances, as shown from study of two-day diets. The allowance for various essentials was met by the following percentages of diets: protein, 85; iron, 78; niacin, 70; vitamin A, 58; thiamin, 54; calcium, 52; ascorbic acid, 33; and riboflavin, 29. Conversely, diets were deficient by more than one-third of the allowance in the following descending order of frequency for specific nutrients: ascorbic acid, 46; riboflavin, 43; calcium, 25; vitamin A, 15; thiamin, 14; niacin, 7; and iron, 4 percent.

It was reported that "only 8 percent of the men had obtained a diet which furnished all nutrients in amounts equal to or greater than the allowances; and not quite 10 percent had diets which furnished 80 to 99 percent of the recommended allowances. Thus, 17 percent of the men had moderately good or excellent diets. The other 83 percent of the men needed some improvement in their diets. Thirty percent of all diets had less than the two-thirds of the allowance for only one nutrient." When the diet was low in only one nutrient, the specific nutrient involved was ascorbic acid in nearly two-thirds and riboflavin in slightly over one-fifth of such diets. In diets below standard in



several nutrients, ascorbic acid was low in nearly two-thirds; riboflavin in 84 percent.

The percentages of 562 white children, 1 to 12 years old, in Philadelphia with diets containing less than the advocated levels of the several constituents were found <sup>35</sup> in 1941-42 to be: niacin, 97; calories, 90; calcium, 71; thiamin, 60; protein, 58; iron, 53; riboflavin, 53; ascorbic acid, 52; and vitamin A, 42. Similarly, the percentages of 38 white adolescents, 13-20 years old, were: calcium, 100; calories, 93; niacin, 86; protein, 71; iron, 71; vitamin A, 71; ascorbic acid, 71; thiamin, 64; and riboflavin, 64, respectively.

Upon inquiry into the diets of 56 white persons, 15 years or older, in Wayne County, North Carolina, in the summer of 1942,<sup>24</sup> it was found that most of the individuals had less than the approved daily quanta of the several essential substances: namely, riboflavin, 100; ascorbic acid, 93; thiamin, 91; calcium, 86, iron, 66; vitamin A, 62; and protein, 55 percent of the persons, respec-

TABLE 6

PERCENTAGES OF PERSONS IN THE UNITED STATES, DISTRIBUTED ACCORDING TO GEOGRAPHICAL SECTIONS, WHO WERE FOUND NOT TO EAT PROTECTIVE FOODS

Types of essential foods	New England and Middle Atlantic	East Central	West Central	South	Far West
Citrus fruits, tomatoes or greens.....	46	41	43	53	41
Dairy products .....	43	31	31	31	28
Leafy and yellow vegetables.....	28	26	25	21	24
Other vegetables or fruits.....	8	8	5	12	7
Meat, fish, and poultry.....	9	12	12	14	14
Whole grain or enriched products.....	3	4	2	3	3

tively. Receiving less than half the advocated daily amounts of the various nutrients were the following percentages of persons: riboflavin, 79; ascorbic acid, 77; thiamin, 43; vitamin A, 27; calcium, 25; iron, 5; and protein, 2.

Of 39 colored persons, the following percentages took less than the advised daily moieties of the several nutrients: riboflavin, 97; ascorbic acid, 92; thiamin, 90; calcium, 82; iron, 79; vitamin A, 72; and protein, 59. Very large percentages of them ate less than half the recommended daily allowances of the various essentials: riboflavin, 82; ascorbic acid, 69; thiamin, 46; vitamin A, 44; calcium, 38; iron, 18; and protein, 10.

A nation-wide canvass in February, 1943, in which one-day diet records were obtained from selective samplings of the population in each of the forty-eight states, showed that the following percentages of persons had none of the respective protective foods:<sup>36</sup> citrus fruits, tomatoes, or salad greens, 45; dairy products, 34; leafy and yellow vegetables, 25; other vegetables or fruit, 8; meat, fish or poultry, 12; whole grain or enriched products, 3. Distributed according to geographical sections, the percentages of persons who did not eat the protective foods are shown in Table 6.

In Tables 7 to 11 are summarized the results from recent surveys, showing the percentages of persons failing to receive the recommended dietary allowances and the extent to which their diets are deficient in the several essentials.

It should be stated that some experts hold that since the recommended



dietary allowances presumably represent "optimum" nutrition with a margin of safety, perhaps many persons who do not have the margin of safety are in no way deficient. In brief they object to labeling diets as deficient which fail to meet recommended allowances. This viewpoint raises two questions: At what levels of their constituent nutrients are diets deficient? What are the meaning and validity of the distinction between minimum and optimum levels?

Several opinions may be cited in support and interpretation of optimum levels as criteria. According to one: "Food plays an important part in determining the internal environment, and differences in this environment, many of which may be too small to be measured by present methods, definitely affect the plane on which physical and mental functioning go on. As far as the immediate or long-term well-being of a person can be improved through dietary betterment, that person falls short of being truly well fed."<sup>37</sup>

TABLE 7

PERCENTAGES OF PERSONS WHOSE DIETS CONTAINED LESS THAN SEVEN SERVINGS A WEEK OF THE RESPECTIVE ESSENTIAL TYPES OF FOODS

Types of essential foods	North Central States, Women College students 1936-40 (16)	Newport, Maine, children 1940 (28)	Maine University Freshmen (girls) 1941 (29)	Burbank, California, aircraft workers 1941-42 (34)
Citrus fruits or tomatoes.....	57	80	19	49
Dairy products .....	29	71	39	33
Leafy and yellow vegetables.....	40	93	44	56
Other vegetables .....	..	71	22	..
Meat .....	14	35	0	1
Whole grain products.....	66	62	69	..

Another states:<sup>38</sup> "There is no reason to doubt, and good reason to believe, that the very wide margin of beneficial increase over absolute minimal need is as valid for human beings as for the experimental animals." Still another writes:<sup>39</sup> "If more were known about the actual vitamin requirements there would be no difference between minimum and optimal requirements, because the optimal presumably is the value which will permit no known pathology to develop and which will maintain the stores of the body at a maximum. Anything less than that could be considered insufficient."

Publications of the past two years confirm the view that the recommended allowances of the National Research Council provide ample margins above the actual needs of normal people for energy, protein, and iron. But for the other nutrients, the levels are none too high or not high enough, according to the intimations in the latest review of the subject.<sup>40</sup>

Nevertheless, Tables 8 to 11 present the proportions of families whose diets fail to meet not only the full recommended levels for constituent nutrients but also 75, 50, and 25 percent of these levels. From this scale anyone can take his choice of levels as a standard. The percentages of diets failing to meet the lower levels are slightly less than those falling short of the full allowances, but they are still very considerable.



TABLE 8

PERCENTAGES OF PERSONS WITH INTAKE OF THE RESPECTIVE DIETARY ESSENTIALS AT LESS THAN RECOMMENDED LEVELS																			
Essential nutrients	Wilkes Barre, Pa. <sup>13</sup>			Lancaster Co., Pa. <sup>17</sup>			New York, N. Y. <sup>18, 19</sup>		Wilson Co., Tenn. <sup>25-27</sup>					Philadel-phia, Pa. <sup>28</sup>		Wayne Co., N. C. <sup>24</sup>			
	748 white children 1-12 yrs., 1937	39 white school adolescents 13-20 yrs., 1937	14 colored children 1-12 yrs., 1937	42 white children 1-12 yrs., 1938-40	20 white adolescents 13-20 yrs., 1938-40	38 white adults, 1938-40	2,037 high school pupils 1939-40	293 private high school pupils, 1939-40	457 white adults, 1941	194 colored adults, 1941	113 white adolescents, 1941	76 colored adolescents, 1941	206 white infants and children, 1941	115 colored infants and children, 1941	Burbank, California, <sup>24</sup> 250 aircraft workers, 1941-42	562 white children 1-12 yrs., 1941-42	38 white school adolescents 13-20 yrs., 1941-42	56 white, 15 yrs. and over, summer, 1942	39 colored, 15 yrs. and over, summer, 1942
Calories	93	100	100	36	30	87	74	68	82	95	89	100	69	95	87	90	93	55	59
Protein	81	100	92	21	20	8	44	26	51	74	78	94	61	87	15	58	71	55	82
Calcium	92	100	100	64	70	24	72	61	61	77	87	93	77	99	48	71	100	86	79
Iron	81	97	92	17	20	10	64	36	59	69	84	89	70	83	22	53	71	66	79
Vitamin A	67	85	54	33	45	37	65	35	45	48	60	59	45	51	42	42	71	62	72
Thiamin	74	100	85	36	45	84	53	18	94	98	93	96	79	93	46	60	64	91	90
Riboflavin	99	100	100	81	90	97	70	36	91	99	85	97	55	91	71	53	64	100	97
Niacin	..	..	..	..	..	..	..	..	..	..	..	..	..	..	30	97	86	..	..
Ascorbic acid	71	95	85	24	30	5	58	20	52	62	68	73	67	70	67	52	71	93	92

TABLE 9

PERCENTAGES OF PERSONS WITH INTAKE OF THE RESPECTIVE DIETARY ESSENTIALS AT LESS THAN 75 PERCENT OF RECOMMENDED LEVELS

Essential nutrients	Wilkes Barre, Pa. <sup>13</sup>	Lancaster Co., Pa. <sup>17</sup>	New York, N. Y. <sup>18, 19</sup>	Chatham County, North Carolina, <sup>20, 21</sup> 1940-41	Wilson Co., Tenn. <sup>22-27</sup>	Burbank, California, <sup>28, 29</sup> aircraft workers, 1941-42	Philadel-phia, Pa. <sup>30</sup>	Wayne Co., N. C. <sup>24</sup>
	748 white children 1-12 yrs., 1937	42 white children 1-12 yrs., 1938-40	2,037 high school pupils 1939-40	293 private high school pupils, 1939-40	457 white adults, 1941	194 colored adults, 1941	562 white children 1-12 yrs., 1941-42	56 white, 15 yrs. and over, summer, 1942
	39 white school adolescents 13-20 yrs., 1937	20 white adolescents 13-20 yrs., 1938-40	33 2,037 high school pupils 1939-40	293 private high school pupils, 1939-40	113 white adolescents, 1941	76 colored adolescents, 1941	38 white school adolescents 13-20 yrs., 1941-42	39 colored, 15 yrs. and over, summer, 1942
Calories	67	7	33	29	50	77	43	50
Protein	35	0	11	7	24	36	6	14
Calcium	69	33	38	32	43	58	39	61
Iron	42	0	29	12	30	44	10	29
Vitamin A	48	10	47	22	32	30	19	43
Thiamin	46	14	22	4	78	51	23	77
Riboflavin	95	55	36	10	75	39	25	95
Niacin	..	..	..	..	..	..	66	..
Ascorbic acid	51	7	36	..	38	..	71	86
	79	85	7	80	50	53	50	80





TABLE 11  
PERCENTAGES OF PERSONS WITH INTAKE OF THE RESPECTIVE DIETARY ESSENTIALS AT LESS THAN 25 PERCENT OF RECOMMENDED LEVELS

Essential nutrients	Wilkes Barre, Pa. <sup>13</sup>			Lancaster Co., Pa. <sup>17</sup>			New York, N. Y. <sup>18, 19</sup>			Wilson Co., Tenn. <sup>20-27</sup>					Philadel- phia, Pa. <sup>28</sup>			Wayne Co., N. C. <sup>24</sup>		
	748 white children 1-12 yrs., 1937	39 white school adolescents 13-20 yrs., 1937	14 colored children 1-12 yrs., 1937	42 white children 1-12 yrs., 1938-40	20 white adolescents 13-20 yrs., 1938-40	38 white adults, 1938-40	2,037 high school pupils 1939-40	293 private high school pupils, 1939-40	Chatham County, North Carolina, <sup>23, 24</sup> 110 white adults, 1940-41	457 white adults, 1941	194 colored adults, 1941	113 white adolescents, 1941	76 colored adolescents, 1941	206 white infants and children, 1941	115 colored infants and children, 1941	Burbank, California, <sup>25</sup> 250 air-rail workers, 1941-42	562 white children 1-12 yrs., 1941-42	38 white school adolescents 13-20 yrs., 1941-42	56 white, 15 yrs. and over, summer, 1942	39 colored, 15 yrs. and over, summer, 1942
Calories	0	5	0	0	0	0	0	0	..	3	12	3	10	5	21	0	0	0	0	0
Protein	0	0	0	0	0	0	0	0	1	0	4	1	4	3	13	0	0	0	0	0
Calcium	7	28	15	0	0	0	1	1	4	3	9	10	15	7	26	0	7	0	0	5
Iron	1	3	0	0	0	0	0	0	0	1	3	3	8	3	7	0	0	0	0	8
Vitamin A	4	18	8	0	0	0	2	1	9	4	17	5	25	5	20	0	0	0	7	21
Thiamin	1	8	0	0	0	0	0	0	17	5	14	3	13	4	6	0	0	0	5	15
Riboflavin	29	54	23	7	5	8	0	0	23	9	32	7	18	6	20	0	0	0	20	39
Niacin	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	0	2	0	..	..
Ascorbic acid	7	15	23	0	0	0	2	0	45	5	13	7	27	7	16	8	3	0	52	49



## DEFICIENCY STATES: THEIR CAUSES, FORMS, AND DETECTION

All these surveys, without exception, revealed similar results: marked prevalence of dietary inadequacies as judged by recommended standards. The evidence is consistent. From all the reports it is evident that very many persons are not receiving the recommended amounts of dietary essentials. Accordingly, the evidence that such a large proportion of the population is eating unsatisfactory diets points to possible widespread prevalence of deficiency states. Indeed, inasmuch as diet surveys have limitations and other factors besides diet enter into the production of deficiency diseases, it is possible, if not probable, that deficiency states are more prevalent than indicated by results of dietary surveys.

Even when the diet seems to contain sufficient amounts of essentials, its adequacy is often apparent rather than real. Its adequacy is usually estimated from calculated values. It is well known that heat markedly destroys vitamin C, and it has recently been demonstrated that light causes destruction of riboflavin in food. Accordingly, analysis of prepared food shows much less than the estimated amounts of these essentials. Usually in estimates of diets allowance is made for loss of vitamin C by heat, but the deduction is often too small. No allowance is made for the destruction of riboflavin by light. Hence, diets adequate by calculation may really be deficient.

Furthermore, surveys at any one time will show only dietary deficiencies at that period. It is probable that many persons having a good diet during the survey previously had a poor diet. Few persons have had a good diet throughout life. Unless a perfect diet has been eaten regularly, deficiency states will not have been prevented and nutrition will not be perfect. Slight deficiencies are apt to be ignored as if they were without effect. Nor must the diet have been unsatisfactory over the entire past to lead to this undesirable state; it may have been poor for a short time or at intermittent periods. Persons who proudly assert that their diet *is* exemplary seem to overlook their past record until prompting reminds them that their diet has been satisfactory only for the last few years, that it was unsatisfactory for a longer or shorter period in the past. They are apt to live in a false sense of security in the belief that the change to a satisfactory diet immediately and automatically expunged the consequences of their past unfavorable record. Kruse has pointed out that time and degree of deficiency are important in the evolution of deficiency states.<sup>41</sup> Deficiencies occurring periodically or persisting over years produce cumulative effects. Slight deficiencies lead to cumulative effects just as truly as marked deficiencies. Partially, indeed slightly, deficient diets eaten regularly and periodically over many years have their consequences. Such conditions are very common. For these reasons, deficiency states are more prevalent and severe than indicated by dietary survey.

Even when diet actually contains the recommended amounts of essentials

for basal needs and the body is functionally sound, deficiency states may still occur. The standards of dietary essentials are average and make no allowance for individual differences and all increased needs. In a review of this topic Jolliffe <sup>42</sup> has pointed out that certain conditions known as conditioning factors raise the requirements for essentials above the basal level. First, sex and age are indices of increased need: the former because of pregnancy and lactation; the latter because of growth. Secondly, physical exertion raises requirements for essentials. When persons with acute pellagra are kept in bed, even though they be given a pellagra-producing diet, their pellagra temporarily disappears. Thirdly, exposure to light has been reported as increasing the need for certain essentials, particularly riboflavin, nicotinic acid, and vitamin A. Fourthly, exposure to toxic substances increases bodily needs for essentials. Alone, any one or all of these several factors do not produce deficiency states but they may exert a determining or occasioning influence.

In setting dietary allowances, the influence of age, sex, pregnancy, lactation, and physical activity is recognized. It is evident that dietary requirements are not constant, fixed, absolute values but are relative to the conditioning factors that increase bodily needs. Often these factors act in combination. Hence, deficiency states may occur on a seemingly adequate diet. Thus, assuming a satisfactory internal mechanism, a deficiency disease may arise from an insufficient intake relative to age, activity, and exposure to light, as well as to other forms of trauma. Of course, if a deficiency state is already present, lowered storage and impaired tissue condition also raise the requirements.

Still other conditioning factors raise bodily requirements for essentials creating a deficiency. For the most part they are diseases, including infections, and therapeutic agents. Differentiation of these conditioning factors from the preceding group is largely artificial. For one thing, they are not regarded as natural events in the sense that disease is not to be accepted as a necessary or normal occurrence. Besides, they are apt to raise the tissue requirements for essentials beyond what food can supply; indeed, in the case of infections, temporarily beyond what the body can utilize. Then too, they are more difficult to control. But they are just as truly conditioning factors as growth, physical exertion, or light. Often all these factors are called the causes of a deficiency state. Accordingly, there are said to be multiple causes of a deficiency disease. More properly, it should be said that there are multiple conditioning factors. They influence the bodily requirements of an essential.

Accordingly, the term deficiency disease should connote a deficiency in the bodily tissue rather than in the diet. Deficiency in the body may arise under many conditions which produce failure in external supply; interference with bodily transport or utilization; destruction or excessive utilization in body, or excessive excretion. It is evident that a bodily deficiency brought about in any of these ways represents a proportionate need. A common cause of bodily deficiency is the inadequacy of the diet to meet even basal needs. But, as Jolliffe <sup>42</sup> emphasizes, conditioning factors likewise exert considerable influence. Like inadequate diet, these conditioning factors may have been operating at many times over the stretch of past years. Few persons have



escaped them. Consequently, a high prevalence of deficiency states is to be expected, much higher than indicated by the results of dietary surveys. All in all, it is clear that many factors contribute to the prevalence of deficiency states. And they determine not only the prevalence but also the acuteness and severity of the deficiency states.

All this evidence points with considerable weight to the widespread prevalence of deficiency states. Yet physicians have insisted that, although they have been looking for deficiency diseases, they rarely or never see them. For some time this has seemed to be an outright discrepancy, but a new concept has now resolved and clarified it. By the notions of deficiency diseases which have prevailed, the physicians have been entirely correct in their assertions. But these notions do not cover actual existing conditions. In speaking of deficiency diseases, physicians have been thinking of the severe acute form. Nearly all animal experimentation has aimed at production of this form. Almost all medical literature bears only on the severe acute form. Until recently none other was recognized. But actually there is relatively little of the severe acute form in this country.

Recently, however, Kruse has demonstrated that there is widespread prevalence of deficiency diseases, not in the traditionally severe acute state but in other forms.<sup>41</sup> He has found that they are very prevalent in a chronic form. Following is a summary of his views based upon gross and biomicroscopic examination. In biological behavior the chronic differs from the acute; whereas the acute is rapid, the chronic is slow in development and in response to treatment. Furthermore, both the acute and chronic processes may be of any intensity from mild to marked. Chronic processes of marked intensity are seen quite frequently in the population. In brief, deficiency diseases have been shown to be widespread in previously unrecognized forms: mild acute, mild chronic, and severe chronic. The acute and chronic processes may be present together. Indeed, not infrequently an acute is superimposed on a chronic condition. Each may be in a different stage and of a different intensity. It should be emphasized that this refers to each kind of deficiency disease. Characteristic tissue changes reflecting form, degree, and stage have been demonstrated in each of four deficiency states. So common, so prevalent, so frequently seen are some of these changes, especially their slighter degrees, that they have been regarded as usual or normal. But their specific location, distinctive and characteristic nature, their reversibility, all attest to their pathological identity as deficiency states.

Time is a factor in the evolution of both the acute and chronic forms; but it is particularly important in the development of the chronic process. In man's life, time is measured by age. Accordingly, the prevalence and severity of the chronic processes increase with age. Most of the chronic changes have hitherto been regarded mistakenly as characteristics of senescence. In reality, however, they are deficiencies developing slowly over a lifetime. These chronic processes may be seen beginning in childhood, even in infancy. The lower the economic level, the more frequently, the more severe, and the earlier in life they occur.

A person's nutritional status today depends on the events of all past days. Upon reflection it is clear that few people have had a satisfactory intake or

escaped adverse conditioning factors every day of their lives. These lapses are reflected in tissue changes and, until recently, attempts were seldom made to correct them entirely. Indeed, many persons continue on an inadequate diet for years. Hence, the tissues show the cumulative changes of long, slowly developing chronic as well as rapidly progressing acute processes. All points considered, Kruse concludes, it is to be expected that the prevalence of deficiency states is high.

Newly developed methods are available to detect these states.<sup>43</sup> Gross and microscopic examination of specific tissues for characteristic changes, a method both sensitive and comprehensive, reveals all forms, intensities, and stages, including early or slight lesions. Just as the pathologist, having exhaustively examined postmortem tissue within the scope of his unaided vision, extended his range of perception and observation to slighter changes by applying the microscope, so the physician may observe the lesser changes in living tissue through the biomicroscope. Analyses of blood or urine to determine the concentration of a nutritional essential or the body's saturation with it yield another kind of information. Blood values, reflecting the character of recent diet, indicate the condition of intake, transport or bodily stores of essentials. Much macroscopic, microscopic, and biochemical evidence obtained from surveys is now available.



## THE PREVALENCE OF DEFICIENCY STATES

Recent estimates of the prevalence of malnutrition have varied so greatly that the Food and Nutrition Board of the National Research Council appointed a subcommittee to evaluate existing evidence on this question. Its report,<sup>44</sup> published in March, 1942, concluded that "some types of malnutrition are strikingly obvious to everyone, some are apparent only to the physician who looks for them and some are vague and elusive even to the careful observer using the most accurate specialized techniques. If the first group alone is counted the prevalence will be recorded as low, almost negligible. If the second group is counted it will be recorded as high. If the third group is included, then the rate will be sufficiently high to occasion genuine concern."

At that time the evidence on the prevalence of malnutrition was treated under three headings: 1) food consumption records or dietary surveys; 2) official death rates and hospital admission figures; and 3) medical assessments of the nutritional status in samples of the population. It was pointed out that these three methods should not be expected to yield identical information. Even when each is as fully developed and as accurately interpreted as possible, the results can only show trends reasonably parallel.

In this review the data will be treated somewhat differently in order to bring the existing evidence on prevalence more into line with the modern concept of malnutrition. Enough time has not yet elapsed to have any considerable volume of data sufficient to classify each specific deficiency into its acute, subacute and chronic forms, each with a subdivision of mild, moderate, or severe, or to indicate whether an acute or subacute has been superimposed on one of the chronic forms. Yet it is probable that in time a chapter on the prevalence of malnutrition will by necessity treat each specific deficiency by recording the prevalence into at least four major subtypes; i.e., mild acute, mild chronic, severe acute, and severe chronic. It is now possible only to record the evidence under the following headings: 1) recorded prevalence of severe acute forms; 2) recorded prevalence from chemical examination; and 3) recorded prevalence of signs, less severe or more chronic than the severe acute form.

The first group represents the severe acute plus the severe acute superimposed on chronic; while the last group represents mild acute and subacute, mild to severe chronic, and mild acute and subacute superimposed on the chronic. The second classification represents recent dietary habits which are reflected in blood and urine chemistry without necessarily indicating the presence or absence of tissue changes.

### RECORDED PREVALENCE OF SEVERE ACUTE FORMS

*Evidence from Official Mortality Rates.* In tracing the average pellagra mortality in a group of Southern States over past years, it has been noted<sup>45</sup>

that up to 1928 it climbed progressively, reaching its peak by 1930. During the next two years it showed a fairly rapid decrease, followed by a gradual decline. After the discovery that niacin (nicotinic acid) is therapeutically efficacious for pellagra, the expected further reduction in the mortality rate of the disease had not materialized by 1941. In part this unfulfillment was attributed to the fact that much of the pellagra was chronic and recurrent.

Evidence based on official mortality rates (Table 12) at best can furnish only information on the prevalence of fatal cases of severe acute malnutrition, and of these only those recognized but in whom therapeutic failures resulted. These rates as published, however, do not furnish a reliable index even of deaths from malnutrition. This is due in part to non-recognition of these and of other nutritional factors causing death, in part to mislabeling and in part to the statistical practice of giving precedence to certain diseases as causes of death over others. It would be interesting to know how much malnutrition is

TABLE 12

DEATHS FROM VARIOUS CAUSES IN THE UNITED STATES REGISTRATION AREA, 1933-40

Cause of death	1940	1939	1938	1937	1936	1935	1934	1933
Pellagra .....	2,123	2,419	3,205	3,258	3,740	3,543	3,602	3,955
Beriberi .....	63	40	42	21	11	7	5	1
Scurvy .....	26	29	30	27	33	30	36	28
Rickets .....	161	143	244	235	270	261	292	339

concealed in the 370,600 deaths recorded in 1938 under the heading "diseases of the circulatory system" or in the 75,431 deaths entered under its subclassification "diseases of the heart, unspecified," or among the 2,569 listed as due to "alcoholism," or to know how much is masked by the nomenclature of "senility," "cirrhosis of the liver," and "psychoses."

Another important factor in evaluating this type of evidence is that the morbidity rates for malnutrition are high in proportion to the mortality. For example, Goldberger and his associates estimated on the basis of a survey in 1917 that there were at least 33 cases of pellagra for each death reported. Sebrell has recently pointed out that this figure is too low, for at that time only pellagra with cutaneous manifestations was recognized. A further factor tending to decrease the ratio of mortality to morbidity in recent years is the improvement in methods of care. Independently, both Sydenstricker and Sebrell arrived at an estimate of 100,000 cases of active pellagra in the United States in 1938.

It is therefore the Committee's opinion that the mortality rates published by the bureaus of vital statistics do not indicate the true death rate from classic deficiency diseases. Even the apparent rising incidence in death attributed to beriberi, from 1 in 1933 to 63 in 1940, suggests this; for it is unlikely that there has been a manyfold increase in fatal thiamin deficiency during this period, and the statistical increase must be ascribed to increasing recognition and better diagnosis. It is thus probable that deaths actually due to malnutrition are many times greater than the mortality statistics indicate.

*Evidence from Hospital Admission Rates.* Pointing out that in a setting of



months or years of an inadequate diet, deficiency disease may be precipitated by even minor illness, Goldsmith determined the frequency with which deficiency states were associated with other clinical diseases among 200 ward patients of all ages admitted consecutively to Charity Hospital, New Orleans.<sup>46</sup> Forty percent had evidence which appeared to indicate definite inadequacy of riboflavin, of niacin, or of both vitamins. An additional 27 percent were considered to have probable deficiency. Thus, 67 percent of the patients examined showed clinical manifestations which indicated an insufficient supply of the two vitamins, the prevalence of riboflavin deficiency being greater than that of niacin. Moreover, 25 percent had signs and symptoms suggesting ariboflavinosis.

The several signs of ariboflavinosis and aniacinosis were noted among the 200 patients with the following frequency: changes in color, texture, and papillae of tongue, 111; thick, pigmented skin over bony prominences, 90; visual disturbances, 80; nasolabial sebaceous plugs, 65; nervousness and irritability, 54; conjunctival congestion, 44; cheilosis, 34; sore mouth and tongue, 25; gross vascularization of cornea, 18; chronic diarrhea, 17; pellagrous dermatitis, 9. Signs of deficiency diseases were present in all persons with hyperthyroidism, cirrhosis of the liver, and chronic alcoholism, and in two-thirds or more of the patients who had infections, diabetes mellitus, carcinoma, and diseases of the gastro-intestinal tract.

Evidence of general prevalence based on hospital record-room figures are subject to the same criticism as the official mortality rates, i.e., incompleteness due to non-recognition, mislabeling and precedence given to certain diseases over others. In addition, two other factors make this index unreliable. The first is that the hospitalized population is not a representative group, for, like persons with the common cold, patients with malnutrition enter the hospital only when sequelae develop; on discharge neither the common cold nor malnutrition but only the sequelae appear as the discharge diagnoses. The second is that certain anatomic lesions of malnutrition may be so prevalent that they are disregarded. This is paralleled by the practice of recording other highly prevalent conditions. For example, in 1938, according to Bellevue Hospital records, dental caries was recorded in only 0.68 percent of its patients, whereas it is common knowledge that the incidence of dental caries in the adult population is near 90 percent.

A recent survey by Krupp<sup>47</sup> of 400 consecutive patients admitted to the clinic wards of Stanford University Hospital disclosed "the incidence of definite vitamin deficiency disease" as 3.1 percent. Adding those showing "possible mild signs of vitamin deficiency" the prevalence would be increased to 7.5 percent. A critical analysis of the criteria used in this study would indicate that they were in the main recording the prevalence of severe acute types of malnutrition as represented by pellagra, beriberi, scurvy and rickets.

#### RECORDED PREVALENCE FROM CHEMICAL EXAMINATIONS

Reliable data from this type of evidence is available for iron (hemoglobin), protein and ascorbic acid. Data from chemical examination of blood, vitamin

A, urinary or blood thiamin, riboflavin and niacin are still in the investigative stage and cannot as yet be used for survey purposes. Evidence of vitamin K deficiency by blood prothrombin estimations involves surveys on special groups such as the newborn or surgical patients which are not generally applicable or significant for population groups.

*Iron Deficiency Anemia.* Statistics on the prevalence of iron deficiency (hypochromic) anemia in the population of the United States are not nearly as complete as the figures available for the British Isles. All surveys, however, show a notably high prevalence, especially in children and pregnant women of the lower-income groups. In Table 13 are listed results of the more recent surveys, indicating findings of anemia in from 1.5 to 85 percent of children, 1.0 to 30 percent in adult males, as high as 68 percent in adult females and 9 to 72 percent in pregnancy. It is recognized that inadequacy of other nutrients, particularly of the B-complex or even thiamin alone, chronic blood loss, malignant conditions and possibly factors other than an inadequate intake of iron may result in hypochromic anemia. However, in studies of large groups, particularly of school children, it is probable that the prevalence of hypochromic anemia is a reasonably accurate index of a dietary inadequacy in iron.

*Vitamin C Undersaturation.* Now that reliable methods are available for the determination of ascorbic acid in the blood, reports of the prevalence of vitamin C undersaturation in population groups are appearing with increasing frequency. Plasma levels below 0.6 mg. indicate less than optimal dietary intakes of vitamin C and levels below 0.4 mg. are considered by most students as definitely abnormal. Williams and Wilder <sup>61</sup> have pointed out "that the normal organism in the basal state has remarkably constant levels of the various constituents of the blood, and that these levels are maintained until there is considerable depletion of stores or until there is disturbance of metabolism. Low levels of vitamins in the blood, therefore, may be evidences of severe rather than mild depletion of stores." On the other hand, plasma levels of 0.6 mg. or more can be consistent with the scorbutic state, for after therapy has been instituted the acute anatomical lesions of vitamin C deficiency, though receding, remain for some time; while chronic scorbutic lesions as represented by hypertrophy or atrophy of the gingival papillae will remain for months. It cannot be too strongly emphasized that low values of ascorbic acid always indicate an unsatisfactory vitamin C balance, but do not indicate the presence of anatomical lesions; similarly normal ascorbic acid values do not rule out vitamin C deficiency as the cause of an anatomic lesion, for recent intake may have elevated the blood level without curing the anatomic lesion.

As shown in Table 14 there is evidence of vitamin C depletion (plasma levels below 0.6 mg.) in a large proportion of our population, varying from 5.5 to 85 percent of different groups. Of special significance is the high prevalence in school children. For example, in small Michigan communities, 49.3 percent of 475 children had serum levels of ascorbic acid below 0.5 mg. percent, and in 1,589 white grade school children in Philadelphia 32.3 percent were below 0.55 mg. percent. In New York City public school children of high school age serum ascorbic acid below 0.4 mg. percent was 32.0, and in private school children only 2.9 percent.



TABLE 13  
PREVALENCE OF SECONDARY (IRON DEFICIENCY) ANEMIA

Locality	Subjects	Criteria gms. Hb. per 100 cc. blood unless otherwise specified	Percentage deficient
Ann Arbor . . . . .	158 pregnant women (urban) <sup>48</sup>	10.0 C.I. 0.9 C. Hb. 26 x 10-12 G	26.6
New York City . . .	a) 325 pregnant women, iron therapy <sup>49</sup> b) 307 pregnant women, no iron therapy <sup>49</sup>	a) R.B.C. 4,000,000 b) Hb. 11.6	a) 28.0 b) 72.0
Oklahoma City . . .	1,000 private obstetric patients <sup>50</sup>	a) R.B.C. 3,500,000 b) Hb. 10.2	a) 16.4 b) 9.2
United States . . . .	pregnant women (review) <sup>51</sup>	"Iron deficiency anemia"	30.00-60.00
New York City . . .	a) 336 private school children <sup>52</sup> b) 425 public school children <sup>52</sup> c) 138 WPA employes <sup>52</sup> d) 198 nurses <sup>52</sup>	Boys, 13 gm.; girls, 12 gm. Boys 13 yrs.: 12.8 to 13.5 gm. 14.0 12.0 12.0	a) 1.5 b) 3.3 c) 34.0 d) 27.0
Pennsylvania . . . .	428 school children (representative economic section) <sup>54</sup>	11.5 10.0	57.0 13.0
Pennsylvania . . . .	2,400 all ages <sup>55</sup> preschool school adult	11.5 10.0 11.5 10.0 11.5 10.0	Male 51.0 ±20.0 14.0 5.0 20.0 ±15.0 Female 56.0 ±20.0 26.0 4.0 30.0 ±15.0
Philadelphia . . . . .	a) 1,589 white 5th grade children <sup>35</sup> b) 179 Negro 5th grade children <sup>35</sup>	12.0 11.5 11.0 12.0 11.5 11.0	29.7 13.9 5.1 69.7 47.4 25.2
New York City . . .	100 hospital patients (medical) <sup>56</sup>	10.2	23.0
Florida . . . . .	620 rural school children (6-10 yrs.) <sup>57</sup>	11.7 9.6 11.7 9.6	85.2 57.2 77.4 33.4
Tennessee . . . . .	1,161 all ages, rural male and female, white and Negro <sup>58</sup> a) 206 white children b) 115 Negro children c) 289 white adult males d) 281 white adult females e) 135 Negro adult males f) 135 Negro adult females	12.5 11.0 12.5 11.0 12.5 11.0 12.5 11.0 12.5 11.0 12.5 11.0	34.6 8.1 47.5 7.7 74.7 15.6 5.5 0.7 28.7 7.4 25.9 2.2 68.8 26.6
Southern California	1,170 male aircraft workers (94 percent being 19 to 39 yrs.) <sup>59</sup>	12.5 11.0	1.03 0.17
Michigan . . . . .	549 children in small communities <sup>60</sup>	14.0 10.0	39.7 9.0

TABLE 14  
PREVALENCE OF ASCORBIC ACID DEPLETION

Locality	Subjects	Criteria mg. ascorbic acid per 100 cc. serum unless otherwise specified	Percentage deficient	
			Autumn	Spring
Maine .....	86 school children <sup>62</sup>	0.6 0.4	69.0 46.0	85.0 63.0
Tennessee .....	380 "normal" children <sup>63</sup>	0.7	Summer 45.0	Spring 61.0
Cincinnati .....	69 orphanage children <sup>64</sup>	0.5 0.25		15.9 5.8
New York City...	a) 342 private school children <sup>62</sup>	0.6 0.4	a) 5.5 2.9	
	b) 425 private school children <sup>62</sup>	0.6 0.4	b) 49.9 32.0	
	c) 165 WPA employees <sup>62</sup>	0.6 0.4	c) 55.2 39.4	
Washington .....	} College students <sup>65</sup>	20 mg. daily excretion	30.0-35.0	
Montana .....				
Oregon .....				
Utah .....				
Massachusetts ...	College students			70.0
Rhode Island ....	College students			85.0
Tennessee .....	1,161 all ages, male and female, white and Negro <sup>68</sup>	0.8 0.6 0.3	45.9 25.6 7.9	
	a) 693 rural white, all ages	0.6 0.4	27.0 14.0	
	b) 370 rural colored, all ages	0.6 0.4	30.0 16.0	
North Carolina...	218 persons, rural mill community <sup>66</sup>	0.3 0	61.0 27.0	
California .....	70 hospital patients (surgical) <sup>67</sup>	0.5 0.3 0.15	77.1 56.9 12.9	
New York City...	100 hospital patients (medical) <sup>68</sup>	0.7	33.0	
Chicago .....	100 hospital patients (unspecified) <sup>68</sup>	0.4 0.3 0.2	38.0 19.0 3.0	
New York City...	157 hospital patients (medical) <sup>69</sup>	0.4	42.0	
Southern California	1,170 male aircraft workers (94 percent being 19 to 39 years of age) <sup>69</sup>	0.8 0.6 0.2	70.2 52.4 10.7	
Michigan .....	475 children in small communities <sup>60</sup>	0.9 0.5 0.1	76.4 49.3 12.0	
Kansas .....	78 normal industrial workmen <sup>70</sup>	0.75 0.50 0.25	84.7 64.1 39.7	
Philadelphia .....	1,589 white grade school children <sup>35</sup>	0.8 0.55 0.3	48.9 32.3 16.5	
	179 Negro grade school children <sup>35</sup>	0.8 0.55 0.3	67.4 46.9 21.2	
Durham, North Carolina	782 young men at National Youth Administration Work Center <sup>71</sup>	0.6 0.3 0	84.9 54.6 9.5	



*Riboflavin.* Feder, Lewis, and Alden <sup>72</sup> have recently surveyed the urinary riboflavin levels in 658 persons comprising laboratory workers, hospital and private patients, medical students, and men called for a physical examination by a Draft Board in Atlanta, Georgia, as well as a large number of individuals from the outlying rural area. From reported data they concluded that perhaps 0.8 microgram of riboflavin per milliliter of urine should rightfully be a critical level above which a value would indicate that the person had an adequate riboflavin status and below which the value would show that the person was to be considered deficient. But to avoid a charge of selecting optimum as the critical level, they set 0.3 microgram per milliliter as the arbitrary level and considered a person with a value below it as deficient in riboflavin. On this basis the following percentages of persons were designated deficient in riboflavin: medical students, 25; white draftees, 30; colored draftees, 50; rural dwellers, 65. It was concluded that widespread riboflavin deficiency prevailed in the area.

*Serum Proteins.* The effects of inadequate protein intake on growth, reproduction, and vigor are well-known phenomena in experimental animals. In man, however, protein deficiency is recognized almost solely by a decrease in the proteins of the blood with the resulting loss of osmotic pressure and the development of edema. The reduction of blood proteins may occur as the result of an inadequate dietary intake but it may occur as a result of the conditioning factors of malabsorption, poor utilization, increased excretion, or physical exertion. The reduction of serum proteins occurs principally in the albumin fraction, the globulin being normal or slightly elevated in uncomplicated cases and reduced only in severe cases. The most common standards used in this country are those of Peters and Eisenman based on the Kjeldahl technic. They are: total proteins, 6.0-8.0; albumin, 4.0-5.5; and globulin, 1.4-3.0 gm. per 100 cc., respectively.

For survey purposes either total protein below 6.0 grams percent or albumin below 4.0 grams percent, more preferably the latter as the albumin fraction is more influenced by dietary factors, may be taken as definitely abnormal. On this basis, surveys of population groups in rural Tennessee and of industrial workers in California, serum albumin below 4.0 grams percent occurred in 1.7 to 21.4 percent of the population groups studied (Table 15). The lowest prevalence was in children with no significant difference found in white and colored. The greatest frequency was found in colored females, being three times the prevalence found in white males or females and almost twice that found in colored males.

#### MEDICAL ASSESSMENT OF NUTRITIONAL STATUS

*Thiamin Deficiency Polyneuropathy.* Though criteria have been established for the diagnosis of mild polyneuropathy none of these signs alone can as yet be said to be due only to a deficiency of thiamin. These signs must be interpreted with a knowledge of the history and of other findings both clinical and laboratory. Nutritional surveys have as a rule neglected to record the presence of such signs as absence of ankle and knee jerks, plantar dysesthesia, calf muscle tenderness and impaired vibratory sensation in the toes. In a

survey by Borsook, Alpert and Keighley<sup>59</sup> of 1,153 male aircraft workers, 94 percent being 19 to 39 years of age, 16.9 percent had one or more of the following signs: absence of ankle and knee jerks, 0.3; absence of ankle jerk, 1.8; calf muscle tenderness, 0.4; plantar dysesthesia, 14.7; and loss of vibratory sensation in toes to C-128 tuning fork, 0.7 percent, respectively.

Confirmation of the frequency of these findings has been observed by Harris, Weeks and Kinde<sup>60</sup> in 769 Michigan school children. Their findings were as follows: absence of ankle jerks, 4.3; absence of knee jerks, 2.2; calf muscle tenderness, 2.6; plantar dysesthesia, 2.3; and impaired vibratory sensation in the toes to a C-128 tuning fork, 8.3 percent, respectively.

TABLE 15  
PREVALENCE OF HYPOPROTEINEMIA

Locality	Subjects	Percentage deficient	
		Criterion total protein below 6.0 gm. percent	Criterion serum albumin below 4.0 gm. percent
Tennessee .....	1,161 rural, all ages, male and female, white and Negro <sup>58</sup>	2.7	8.0
	206 white children	1.4	1.9
	115 Negro children	5.2	1.7
	289 white adult males	2.0	7.9
	281 white adult females	2.8	7.1
	135 Negro adult males	2.9	12.8
	135 Negro adult females	2.9	21.4
Southern California .....	195 aircraft workers (94 percent being 19 to 39 years of age) <sup>59</sup>	...	2.6

In surveys it is not possible to categorically state that all these abnormal neurological signs are due only to thiamin deficiency, as other deficiencies and causes other than malnutrition will produce these signs. In this age group—school children and young healthy adults—other causes, such as neurosyphilis, pernicious anemia, and arteriosclerosis, must have been very infrequent.

If these signs in only half the subjects are eventually proven to be the result of a thiamin deficiency, the prevalence of neurologic changes on a thiamin deficiency basis is large enough in school children and in adult males under 40 years of age to cause genuine concern.

*Riboflavin Deficiency.* Cheilosis, angular stomatitis, characteristic seborrheal skin lesions and significant degrees of superficial corneal vascularity are all signs which have been attributable to riboflavin deficiency. The most frequent of these signs is corneal vascularity which is undoubtedly associated with riboflavin deficiency in many subjects.<sup>73-75</sup> In acute cases this condition can be observed by slit-lamp biomicroscopy to disappear following riboflavin administration, return when riboflavin is withheld and disappear again when treatment is resumed. In this acute type of lesion the ocular symptoms of burning, itching, asthenopia, photophobia and lacrimation are extremely common. Several investigators<sup>52, 53, 58-60, 76</sup> have used corneal vascularity as an index of riboflavin deficiency in surveys of population groups. Its validity for



this purpose has been questioned, for many <sup>77-79</sup> consider that it is not yet confirmed that all or nearly all of these vascular changes are due only to riboflavin deficiency or that they are a necessary accompaniment of other undoubted signs of riboflavin deficiency. The consensus of this group at the present time seems to be that one cannot assume that all significant degrees of corneal vascularization are due to riboflavin deficiency. Sandstead,<sup>80</sup> following riboflavin therapy for 60 to 110 days, found no significant change in corneal vascularity which could be ascribed to this therapy. He concludes "at the present time, therefore, it seems doubtful that superficial vascularization of the cornea, as observed in this study and as found in the general population, should be considered a diagnostic sign of riboflavin deficiency."

On the other hand, Tisdall *et al.*<sup>81</sup> found the prevalence of corneal vascularization among apparently healthy young adults in Canada surprisingly high and seeming to vary with the riboflavin-containing foods in the diet. Riboflavin in large dosage for a period of two months decreased vascularization of the cornea in a large percentage of cases and caused marked improvement in disturbing ocular symptoms. Youmans and Patton<sup>82</sup> have observed a considerable number of subjects with mild but definite corneal vascularization not accompanied by ocular symptoms and in whom there was no correlation with dietary intake of riboflavin, other dietary factors or other evidence of deficiency disease. Reexamination of these subjects showed an improvement or disappearance of the vascularization in the winter or spring season compared with the fall, in spite of a lower intake of riboflavin in the former period. It was suggested that factors besides riboflavin are concerned. Youmans and Patton<sup>82</sup> concluded "that corneal vascularization, even of the characteristic type and of definite degree, does not always mean a general riboflavin deficiency." They then point out that under these circumstances the lesion loses some of its value as a diagnostic test of riboflavin deficiency in population groups. But for clinical purposes they point out that significant degrees of corneal vascularity, plus the history and the results of a therapeutic trial, are not only the most helpful and reliable but the simplest and quickest means of detecting mild riboflavin deficiency.

Pertinent to this view is the rôle of conditioning factors. Sandstead<sup>80</sup> points out the possibility of the acute exanthemata playing a rôle in the high prevalence of corneal vascularity. It will be recalled that physical exertion, and light have been cited as examples of conditioning factors in relation to intake. The fact that physical exertion may induce nutritional edema is not regarded as impairing the diagnostic sign of edema in that deficiency state. Nor is nutritional edema regarded as being "caused" by exertion. Just as physical exertion may precipitate nutritional edema, and light, trauma, heat, dirt, or filth may induce or intensify pellagrous skin lesions, so corneal vascularity may be conditioned by light.<sup>75, 83, 84</sup> But this does not affect the validity of edema, of the characteristic skin lesion, or of corneal vascularity as diagnostic signs of protein, niacin, or riboflavin deficiency, respectively. Neither does it necessarily question the fundamental relation of protein, niacin, and riboflavin to these respective deficiency states.

Another point should be emphasized. It has been reported that recession of the lesions of chronic ariboflavinosis is very slow and is complete only after a very long period of time.<sup>41</sup> This is likewise true of other deficiency states. Therefore, short periods of therapy without apparent results may lead to erroneous conclusions. In none of the cases where no improvement has been reported was therapy continued more than a few weeks or a few months.

By use of corneal vascularity as a criterion of ariboflavinosis, but with recognition of the possibility that it may give too high a figure, the apparent prevalence of ariboflavinosis (Table 16) in children varies from 2.3 percent in high-income families to 75 percent in low-income families, while in adults its prevalence seems to vary from 20 to 40 percent.

TABLE 16  
PREVALENCE OF CORNEAL VASCULARITY (ARIBOFLAVINOSIS)

Locality	Group	Corneal invasion	Percent
New York City...	354 high school students high-income families <sup>52</sup>	Any degree of corneal invasion	2.3
	495 high school students low-income families <sup>52</sup>	Any degree of corneal invasion	75.8
	138 WPA employes <sup>52</sup>	Any degree of corneal invasion	38.4
	198 nurses <sup>53</sup>	Any degree of corneal invasion	47.0
Southern California	1,171 male aircraft workers (94 percent being 19 to 39 years) <sup>59</sup>	"Streamer type"	42.5
		Significant invasion	20.9
Tennessee .....	110 white—all ages <sup>58</sup>	Significant invasion	0
	47 Negro—all ages <sup>58</sup>		
Michigan .....	710 school children <sup>60</sup>	More than three arcades	53.9
Philadelphia .....	1,960 grade school children <sup>76</sup>	Any degree of corneal invasion	50.3
		Invasion of limbus only	39.3

*Vitamin A Deficiency.* Xerosis conjunctivae is a lesion characteristic of vitamin A deficiency in animals <sup>85, 86</sup> and one that is reported to respond to vitamin A therapy in man.<sup>87</sup> From this it should not be inferred that all "spots" or elevations appearing on the eyeballs peripheral to the cornea are xerosis conjunctivae. Some are, but others are pterygium, and others scleral deposits of varying composition. The contention of Kruse <sup>87</sup> that xerosis conjunctivae is a manifestation of chronic vitamin A deficiency has been criticized by Berliner <sup>88</sup> who points out that not all the photographs published by Kruse were characteristic of xerosis conjunctivae, that the vitamin A blood levels are not low, and that many of the lesions in question are those usually ascribed to simple senile changes. It should be noted, however, that trained ophthalmologists after gross and slit-lamp examinations frequently disagree on the appropriate anatomic labeling in a high proportion of "spot" cases. The blood level of vitamins does not necessarily reflect the state of the tissues and cannot be used as a criterion for the absence of a deficiency disease, particularly



a chronic deficiency anatomically manifest.<sup>89, 90</sup> In this type of malnutrition the blood reflects the recent intake of vitamin A. Also, to designate lesions of the conjunctivae as senile changes with the implication that senility *per se* is responsible for them is unsatisfactory.<sup>41, 91</sup> This merely identifies the condition with a population in which it is most frequently found, but does not explain its etiology. Even in arteriosclerosis, age is not the essential cause. As William Boyd<sup>92</sup> has stated, "The advance in years merely permits some slowly acting cause to produce the effects in the vessels." In discussing the lesions of chronic malnutrition, Kruse states:<sup>41</sup> "Not all elderly persons show the changes. On the other hand, they occur in children. Time, not senility, is the essential point, and time does not start the changes, it is simply a dimension over which they progress. They are specific avitaminoses in a state of chronicity, due usually to respective dietary deficiencies running over a period of years. Their prevalence and severity vary with the number and degree of deficient diets. . . . Most important of all, they are reversible, yielding slowly but completely to appropriate therapy." This latter point has been partially confirmed by Jolliffe and Stern<sup>93</sup> who observed that of ten subjects given 50,000 units of vitamin A twice daily by mouth for 10 months, seven showed evidence of responding in the manner described by Kruse. It seems therefore that some of these lesions respond to vitamin A therapy. Some may not, but sufficient time has not yet elapsed to decide this point definitely.

Following a twenty-five week period of high intake of carotenoids (200,000 I.U. of vitamin A as carotene) and a ten week period on 10,000 units of carotene, Getz<sup>93a</sup> has subsisted during the past one and a half years on a diet providing less than 500 I.U. vitamin A daily. At intervals during the entire period of deprivation his conjunctivae were observed with the biomicroscope by several observers; his visual adaptation was tested and his plasma vitamin A concentration was determined. The first change noted during the period of vitamin A deficiency was the appearance of conjunctival changes described by Kruse. Several months later some visual dysadaptation developed. The conjunctival changes were first seen in the third month on the low vitamin A diet, progressing and becoming grossly visible; dysadaptation began in the seventh month; and scaliness on the elbows was observed in the eleventh month. But the level of plasma vitamin A, reaching 180 I.U. per 100 ml. during the preliminary period of high vitamin A intake, never did fall below 93 I.U. during the deprivation period. Not only were his vitamin A stores presumably well stocked at the beginning of the deprivation period, but also conditioning factors during the period were in operation only at a minimum. So far as is known, this is the first attempted and reported instance of experimentally induced conjunctival changes in a person as a result of vitamin A deprivation.

On the basis of "gross" spots the prevalence (Table 17) varies from 7 to 60 percent of the population groups studied, while biomicroscopic changes occur in 50 to 99 percent of all subjects. The prevalence of gross spots is higher in males than in females, in colored than in whites, and in older than in the young.

*Vitamin D Deficiency.* With the development of knowledge of the cause and prevention of rickets, the prevalence of severe rickets has been reduced greatly in recent years. Twenty years ago, except for regions blessed with more than average sunshine, the prevalence of clinically detectable rickets in infants was estimated by Hess <sup>94</sup> at about 75 percent. Present figures indicate that the prevalence of active and healed clinical rickets is approximately 20

TABLE 17

## PREVALENCE OF CONJUNCTIVAL CHANGES (AVITAMINOSIS A)

Locality	Group	Criteria	Percent
New York City...	494 high school students from low-income families <sup>52</sup>	Gross spots	7.7
		Biomicroscopic changes	86.6
	143 WPA employes <sup>52</sup>	Gross spots	45.5
		Biomicroscopic changes	99.3
Southern California	198 nurses <sup>53</sup>	Gross spots	34.0
		Gross spots	34.0
	1,172 male aircraft workers (94 percent being 19-39 years) <sup>59</sup>	Gross spots	47.6
		Biomicroscopic changes	96.6
Michigan .....	710 school children <sup>60</sup>	Opacity	2.5
		Significant biomicroscopic changes	48.2
New York City...	549 psychiatric patients <sup>69</sup>	Gross spots	60.0
Philadelphia .....	1,239 grade school children, Part I <sup>76</sup>	Gross spots in characteristic location	8.15
		Gross spots in characteristic location	7.39
	473 grade school children, Part II <sup>76</sup>	Elevated spots in non-characteristic locations, exclusively	17.25
		Combination of characteristic and non-characteristic spots occurring simultaneously	2.60
		Biomicroscopic changes:	
		Right temporal region	88.6
		Right nasal region	94.9
		Left nasal region	96.6
		Left temporal region	90.0

percent among children of preschool age. This figure varies greatly from community to community, depending on urbanization, age, season of the year, race, and the practice of preventive treatment.<sup>95</sup> In Newark, New Jersey, Levy and Silver <sup>96</sup> studied, in the spring of 1937, 100 consecutive infants less than 9 months of age and found clinical evidence of rickets in 37 percent.

In North Dakota the state health department reported in 1937 a study of 5,227 preschool children in whom by clinical methods active or healed rickets was found in approximately 20 percent. In 1941 Rhoads and her co-workers <sup>97</sup> reported a study of 233 children at 2 years of age who had received 110 to



1,500 International Units of vitamin D daily for the previous 22 months of life; active rickets was found in 7.7 percent and healed rickets in an additional 6 percent. The prevalence of rickets in 487 autopsied children between 3 and 19 months, dying in a hospital, was found by Follis, Jackson and Park<sup>98</sup> at 48.4 percent.

*Niacin Deficiency.* With a single exception, evidence of this deficiency has been based on the severe acute lingual manifestations. It is obvious that both the acute severe form as manifested by a scarlet red glossitis, and the chronic form as represented by complete papillary atrophy, represent end results that are preceded by changes representing deviations from normal up to these recognized end stages. In the acute form hyperemia and hypertrophy of the fungiform papillae impart first a stippled and later a strawberry aspect before the complete scarlet red glossitis is evident. In the chronic form the stages are hyperemia associated with loss of substance as manifested by fissures, crevices and areas of progressive lowering of the papillae until a completely bald tongue is produced. With these changes as criteria, and their detection by the bionicroscope, all 49 subjects in a study<sup>99</sup> showed some abnormal lingual changes. Upon examination of 198 nurses of New York City, 20 percent were found to have subacute changes characteristic of niacin deficiency.<sup>53</sup>

*Ascorbic Acid Deficiency.* As with niacin deficiency, anatomical evidence of ascorbic acid deficiency has been based on the severe acute gingival lesions. It is also obvious that the severe acute form must be preceded by gingival changes of a degree less than gingivae, consisting of bags of blood which almost completely hide the teeth. In the development of this acute process the first stage consists of capillary engorgement of the interdental papillae and later of the marginal gingivae. As this microscopic lesion advances it can then be seen grossly as a serrated red line marking the interdental papillae and later the marginal gingivae. Then as the lesion progresses the gums become swollen, until eventually the typical scorbutic gums are seen. The chronic form may start as the acute, but somewhere in its development, probably because of the presence of dietary vitamin C but in amounts insufficient to cure completely, atrophy begins as evidenced by pitting on the interdental papillae. It finally leads to gum retraction with formation of calculi between the gums and the teeth, then superimposed infection and so on *ad infinitum* to the end stage of chronic scurvy as represented by complete retraction of the gums, exposing the alveolar surfaces of the teeth, alveolar loosening, infection and extrusion of the teeth. In 49 subjects examined in a study,<sup>100</sup> all showed some degree of these deviations from perfection. In a later study<sup>53</sup> on 198 nurses of New York City acute or subacute changes were observed in 56 percent. The prevalence of undiagnosed scurvy in 487 autopsied children between 3 and 10 months, dying in a hospital, was found by Follis, Jackson and Park<sup>98</sup> at 11.7 percent.

#### DEFICIENCY STATES VERY PREVALENT IN THE NATION

All the evidence is in agreement that deficiency states are common among the population of the United States. Most of them are not the severe acute type.

Rather, they are less intense in degree and very much slower in their course. Predominantly the deficiency states here are mild, moderate or severe chronic forms. Because of their slow gradual development, their presence is commonly unsuspected. In frequency and severity they increase with age and with lowered economic level. As yet optimum nutrition throughout the nation has not been achieved; on the contrary, deficiency states are present on a large scale.



## SIGNIFICANCE OF DEFICIENCY STATES AND OPTIMUM NUTRITION

The serious health handicaps resulting from the florid forms of the deficiency diseases are too well known to require review.<sup>95, 101, 102</sup> Although the severe acute types, with their obvious clinical manifestations, are seen in many parts of the world, relatively few such cases (although perhaps significant numbers) occur regularly in the United States.<sup>44, 47, 103-105</sup> Instead, most of the deficiency states in this country are milder in degree and chronic in type. These various forms constitute a wide zone between obvious deficiency disease and optimum nutritional state. The evidence on the relation of deficiency states in that zone to health is not nearly so clear and has led to some differences in conclusions.<sup>41, 44, 70, 106, 107</sup> Most of the controversy arises out of the confusion of definition, but some of it is based on the type of evidence and its interpretation.

Much of the scientific work in support of the importance of optimum nutrition to health loses its strength if the latter term is defined in the narrow sense as "absence of obvious disease," but becomes distinctly significant if one recognizes health as having more positive attributes. In this latter sense, health has quantitative characteristics involving efficiency, reserves, and the capacity not only to avoid disease but to attain maximum inherited potentialities. This point of view is the basis of preventive medicine and modern public health practice. It is with this definition in mind that the following evidence should be considered.<sup>108-112</sup>

So many factors other than food can enter to influence health during the course of studies on human beings that the full significance of the relation of food to well-being is most easily shown by the use of experimental animals. There are obvious advantages in making observations on a species kept under well-controlled conditions which can be varied at will and can be continued through a generation or several generations if desired. Furthermore, since recession of lesions in chronic deficiency states occurs slowly and requires a long period of therapy, many studies in man on the relation of nutrition to health must necessarily be of long term. Here the use of experimental animals in orientative investigations offers a real economy in time. By this procedure of experimentally searching out the significant data, the subsequent studies on humans becomes less arduous and more straightforward.

Sherman and colleagues<sup>113-114</sup> have shown by a series of carefully planned and executed experiments, extending over a period of years, that diets adequate for the rearing of 50 generations of apparently normal rats can be improved with betterment to the animals. These benefits are made evident by increased growth and efficiency, decreased death rate and increased vitality at all ages. Also, the better-nourished animals not only live longer on the average, but most important of all, the period of prime of life is distinctly increased. These conclusions are based on data of unquestionable statistical

significance. More detailed experiments <sup>115-118</sup> reveal that several chemical factors, i.e., vitamin A, calcium, riboflavin, etc., contribute to this improved health record and that up to four times the adequate quantities of these substances led to increased benefits.

Waterman and Ammerman <sup>119</sup> have likewise shown that increased thiamin beyond that amount judged as distinctly adequate will yield improved growth in rats. The farmer has known for years that there was a difference between "just adequate" and optimum feeding. An increased biological efficiency reflected in superior production of eggs, milk, wool, and meat, material results which can be evaluated in dollars, makes a convincing demonstration of the profitableness of better feeding.<sup>120</sup> On purely biological grounds, one would expect analogous benefits to man as measured in better health performance.

McCarrison,<sup>121, 122</sup> struck by the contrast of the well-developed, healthy appearance of the people of northern India compared to the poor physique, low power of endurance and resistance to disease of those races in the eastern and southern parts of the country, undertook a study of the cause of this difference. After considering other factors, such as race, heredity, climate, and endemic disease, he concluded that the nature of the diet was likely the most important factor. To confirm this conclusion experimentally, he raised identical groups of rats on diets representative of the various regions, and he found the same great contrast in growth, development, and well-being of the animals as was observed among the people.

More recently Radhakrishna Rao <sup>123</sup> has shown that monkeys maintained on diets based largely on milled rice and containing supplementary foods in small quantities, similar in composition to those consumed by the poor rice-eaters in India, show the same poor development and occurrence of gastrointestinal, skin, eye, and mouth lesions as the people in this region.

While these animal studies are strongly suggestive, they are not necessarily proof that man will or does react in a like manner. Orr and Gilks <sup>124</sup> conducted a study to test this point. These investigators compared the physique and health of two African tribes living side by side, alike in most respects except dietary habits. The Masai are pastoral people and consume quantities of milk and meat; while the Kikuyus live for the most part on cereals with some tubers and legumes. The latter tribe is greatly inferior in physical development. The Masai men are on the average three inches taller and 23 pounds heavier, and the Masai women, three inches taller and 27 pounds heavier than the Kikuyu men and women. There was a similar striking difference in the prevalence of chronic respiratory disease, parasite infestation, and general health records. Added evidence that these differences were due to nutrition was indicated by the improvements resulting from changes made in the dietary of the Kikuyu.

#### EFFECTS OF PROPER DIET ON GROWTH AND DEVELOPMENT

Corry Mann <sup>125</sup> also reports observations from an English institution for boys which bear on this point. One group of boys, six to 11 years old, were maintained on their regular diet, which was well-planned and judged to be



good; while other groups received various supplementary foods. The most striking difference observed over a three-year period was between the control group and those receiving an extra pint of milk daily. During the first year the former group gained on the average of 3.85 pounds and 1.84 inches, while those receiving the extra milk gained on the average of 6.98 pounds and 2.63 inches. Orr <sup>126</sup> and Leighton and McKinlay <sup>127</sup> demonstrated the same point on even larger groups. Similar gains in growth and improvement in general health records have been reported both in this country and England as a result of the supplementary feeding through school lunches. <sup>128, 129</sup>

The high percentage of men rejected from the Army because of inferior development and related causes has been distressing to those interested in public health. <sup>130</sup> It is unfortunate that time and facilities have not allowed for better study of the problem in this country. An important experiment in this connection was conducted in England. <sup>131</sup> A group of several hundred young recruits rejected because of under-development and other defects usually considered as due to malnourishment were placed in a camp under optimum conditions of nutrition, exercise, and rest. After several months of such treatment, 87 percent of a group of 834 passed the physical tests which they had previously failed.

Added indirect evidence comes from a recent study <sup>132</sup> of 78,000 Toronto school children which confirms the results obtained many times previously <sup>133, 134</sup> that environmental factors affect height and weight. In this analysis striking differences have been found between the height and weight of children from low-income families and those from higher-income groups. In the Toronto study the percentages of male children under average height in various groups classified according to the occupation of the father were: 29.9, laborers; 24.5, factory operators; 19.8, clerical workers; 18.9, commercial workers; 17.2, owners and managers; and 13.2, professional workers. Thirty-three percent of boys from families on relief were under average height against 22 percent of those not on relief. Only 12.2 percent of those boys attending 10 schools located in prosperous districts were under average height compared with 31.3 percent of those attending 10 schools located in poor districts. Corresponding differences were also found for girls. It is not implied that stature is the only or the most crucial measure of well-being. Nor is it certain that the difference in the quality of nutrition is solely responsible, because differences in sanitation and rest habits also exist between these two groups. However, sound consideration of all the facts certainly implicates nutrition as the most likely cause of this stature effect.

#### EFFECT OF PRENATAL DIET ON CHILD-BEARING

Ebbs, Tisdall and Scott <sup>135</sup> have investigated the relation between prenatal diet and subsequent obstetrical history of 400 women from a low-income group in Toronto. One group found to be on a poor diet was left as a control. A second group on a poor diet was supplied supplementary food during the final trimester of pregnancy, and a third group found to have a relatively good diet was given education in the best use of food. The past obstetrical history,

the various complications of pregnancy, the obstetrician's general rating, ease of labor and birth, breast feeding, response of the child to feeding and infections during the first six months of life, and other factors were measured or evaluated.

They summarize their findings and conclusions thus: "During the whole course of pregnancy, the mothers on a good or supplemented diet enjoyed better health, had fewer complications and proved to be better obstetrical risks than those left on poor prenatal diets. The incidence of miscarriage, still births and premature births in the women on poor diets was much increased. The incidence of illness in the babies up to the age of six months and the number of deaths resulting from these illnesses were many times greater in the poor-diet group. While it is recognized that there are other important factors in the successful outcome of pregnancy, this study suggests that the nutrition of the mothers during the prenatal period influences to a considerable degree the whole course of the pregnancy, and, in addition, directly affects the health of the child during the first six months of life." While more cases are desirable and statistical treatment important, this study presents a strong argument for the importance of optimum nutrition in prenatal care.

Another study along this line has been reported by a Committee of the Peoples League of Health in Great Britain.<sup>136</sup> On the basis of a preliminary survey, a supplement containing those minerals and vitamins found most likely to be short in the diet was given to 1,530 primiparas, while 1,512 continued their regular diet without supplement. The complications of pregnancy, pre-eclampsia and toxemia were statistically less in the supplemented group. The incidence of premature births was also reduced. No differences in birth weight, sepsis after child birth, or character and duration of labor was noted.

#### EFFECT OF AVITAMINOSES ON WELL-BEING AND EFFICIENCY

The recent, well-controlled human experimental studies at the Mayo Clinic<sup>137</sup> add much to our knowledge of the symptoms and handicaps of early mild thiamin deficiency. Subjects kept on diets as adequate in thiamin as many American diets soon developed evidence of fatigue, irritability, muscular tenderness, and mental confusion. These conditions were paralleled by a definite decrease in efficiency in the relatively simple operations about the household. Although these symptoms and discomforts are admittedly not specific to thiamin deficiency, there seems no doubt in this case that thiamin deficiency is the cause. The symptoms were rapidly alleviated in those who received thiamin but continued to progress in those getting the placebo.

There are a number of mild symptoms and lesions of rather vague origin which occur quite commonly in the general population and which, although not particularly serious, are uncomfortable and distracting and must have a cause. Among these are soreness of the mouth and tongue with change in the papillae; swollen and bleeding gums; mild conjunctivitis with lacrimation; burning and itching of the eyes. These are seen in certain deficiency states. The mouth and tongue lesions of niacin deficiency, the gum lesions of ascorbic acid deficiency, and the eye lesions of ariboflavinosis are examples. Recent



efforts <sup>99, 100, 138-140</sup> to observe more closely the changes in these tissues, especially in connection with specific therapy, indicate that many of these cases are due to deficiencies. Conditioning factors in relation to intake undoubtedly may also be instrumental in inducing these lesions and symptoms.

There is a great need for well-controlled studies on the relation of nutritive state to physical and mental efficiency, especially in mild deficiencies. The magnitude of the problem, plus the need for more objective methods have been deterring factors. Several recent attempts in this direction have been reported.

Keys and Henschel <sup>141</sup> have studied the effects of the addition of large daily supplements of thiamin, riboflavin, nicotinic acid, pyridoxine, pantothenic acid, and ascorbic acid to the regular U. S. Army garrison rations on the muscular ability, endurance, resistance to fatigue, recovery from exertion, and certain physiological mechanisms of a group of trained recruits expending 3,700-4,200 calories per day. Test periods of 4-6 weeks, alternating the supplements and placebos, were used. No improvements in performance were observed, which would appear to indicate that healthy young men on an approved ration calculated to contain 1.7 mg. thiamin, 2.4 mg. riboflavin, and 70 mg. ascorbic acid are not demonstrably benefited by a supplement of vitamins B complex and C over a relatively short period of time.

Foltz, Ivy, and Barborka <sup>142</sup> likewise found no influence on recovery from fatigue or increase in muscular efficiency by parenteral injections of vitamin concentrates in persons already on an adequate diet. These studies were interpreted as showing that supplementing an already adequate diet with vitamin B-complex for several weeks has no influence on work output, recovery, or other manifestations of physical exertion. It has been suggested that it would be desirable to have longer experimental periods in such studies.

Simonson, Enzer, Baer, and Braun <sup>143</sup> have reported a study on the influence of vitamin B-complex surplus on the capacity for muscular and mental work. Measurements on endurance in dynamic and static work, recovery of work capacity and pulse rate, maximum muscular force, fatigue of voluntary effort, maximum frequency of finger movements, errors in calculation tests, and fusion frequency of flicker were made on 12 apparently normal subjects who received over a 6-12 week period daily supplements of 6 mg. thiamin; 80 mg. nicotinamide; and 8 mg. riboflavin. The performance of this group was compared with a control group of 11 similar subjects. No effects of the supplementary vitamins were observed in the muscular work tests. However, there was observed an increase in the fusion frequency of flicker in 8 of 12 of the supplement group after 3 weeks, as compared with no change in the placebo group. This increased ability to detect flicker frequency, reputedly "a measure of central nervous system fatigue," dropped to the initial values 4-6 weeks after cessation of the vitamin intake.

When men are restricted to a diet deficient in the vitamin B-complex, the effects on physical performance are striking. Egana, *et al.* <sup>144</sup> have made observations and measurements on 7 healthy physicians who subsisted on a diet deficient in B-complex for 4 weeks and then had small added amounts of brewers' yeast for 2 weeks. Mild and vague symptoms of pain, fatigue, and loss of ambition were observed, and there was a moderate deterioration in

the subjects' physical fitness for exhausting exercises and particularly poor recuperative powers between repeated periods of exhausting exercise on a treadmill. The same changes were rapidly reversed upon the addition of yeast to the diet.

Even more striking were the effects of vitamin B-complex deficiency in men doing daily hard work. A similar study by Johnson, *et al.*<sup>145</sup> from the same laboratory on 10 subjects engaged in hard physical labor showed that under these conditions symptoms of muscular and joint soreness, fatigue, and physical deterioration were more rapidly produced, being noticed after one week on the deficient diet; and while the addition of thiamin improved these conditions, especially muscular symptoms, the addition of yeast was necessary for full recovery.

In trained subjects restricted to diets containing about one-third of recommended daily amounts of the vitamin B-complex, Barborka, Foltz, and Ivy<sup>146</sup> found that subjective symptoms of easy fatigue, irritability, lack of pep, anorexia, and increased leg pain appeared during work periods and work output decreased. All the symptoms completely disappeared and work output returned to normal or better within a few days following supplementation with vitamin B-complex.

It seems probable from the above reports and other evidence that there is a level of nourishment which, while it is not low enough to lead to easily recognized symptoms of deficiency disease, nevertheless does lead to decreased physical and mental efficiency. However, it is evident that additional well-controlled experiments on subjects whose initial nutritional status is known, who can be maintained for long periods on various levels of intake of the several factors (vitamins, proteins, etc.) and whose performance is measured in an objective manner, are needed before satisfactory conclusions can be made regarding nutritional status and physical and mental efficiency.

#### DIET AND DENTAL CARIES

The cause and prevention of dental caries is one of the most puzzling and difficult problems in public health today. There are several schools of thought on the subject, each with its pet theory. The literature is extensive, confusing and controversial.<sup>147-149</sup> It seems evident that the problem is more complex than generally realized, that many factors are probably involved, and that the solution is not in sight.

However, in all this confusion, there are certain observations and conclusions related to nutrition that seem worthy of emphasis. There is sufficient work of good caliber to indicate that attention to diet can not only decrease the prevalence of caries, but give some protection against the further development of that already present.<sup>147, 150-154</sup> Also, it is equally evident that malocclusion, uncleanliness, bacteria, too many sweets and unknown factors are involved in the process.<sup>147-149</sup> Unfortunately, a satisfactory experimental animal has not been found for this type of work.

Somewhat over a decade ago it was shown by Mellanby<sup>155</sup> that dogs reared on diets faulty in calcium and phosphorus, as well as vitamin A and/or D,



lead to poorly calcified (hypoplastic) and otherwise malformed teeth and supporting structures, factors said to be important in the caries problem. Subsequent well-controlled studies by the same author in a London institution for children<sup>154, 156</sup> clearly showed that the prevalence of hypoplastic teeth and caries could be greatly decreased by the addition of the above factors. Confirmatory observations have been reported by several others. Boyd and Drain<sup>152, 153</sup> and Howe and White,<sup>150, 151</sup> and others have shown that the increased use of meat, milk, fruit and vegetables in the diet is followed by a reduction in occurrence and severity of caries. The formation of a hard, secondary dentin as a protective reaction at the base of developing caries as a result of better nutrition is reported by several investigators.<sup>150, 151</sup>

One of the most interesting facts about dental caries is the geographic distribution of high and low prevalence groups.<sup>157</sup> In central Alaska, certain island groups and other small areas of the world usually remote from modern influence, the prevalence of dental caries is conspicuously low; while in most other areas it is 95 to 98 percent.<sup>158, 159</sup> When members of these low-caries groups come in contact with world commerce, the prevalence of dental caries increases rapidly. This has been observed repeatedly. The report of King<sup>160</sup> gives a full bibliography of these observations, as well as details of his own studies in the Island of Lewis in the Outer Hebrides off the Scottish Coast. The prevalence of dental caries in a large group living on the coast near an active port was about like that observed in London, e.g., 98 percent; while inland where modern influence has not reached, the frequency was on the average about 40 percent. The other most conspicuous difference in these two areas was a change of diet from the natural foods of high nutritive quality of the interior to that containing highly-processed flours, sugar, etc.

Thus, there is much presumptive evidence indicating that nutrition plays a part in the dental caries. The question is how? Bunting and his associates<sup>147, 161</sup> contend from their evidence that it is the presence of high carbohydrate in the diet with subsequent bacterial action. But the evidence indicates in addition that positive nutritive qualities of the diet also are involved.

#### NUTRITIONAL STATUS IN RELATION TO SUSCEPTIBILITY AND RESISTANCE TO INFECTIONS

Considering the variety of infectious agents studied, the different routes and extent of exposure employed, the many types, combinations, and degrees of deficiencies involved, and many other factors which are sure to enter any observations on the relation of nutrition to infection, it is not surprising that the literature on this subject appears inconsistent, confusing, and difficult to evaluate.<sup>162-164</sup> All infectious processes are not the same. Each shows specific characteristics. Nor do the various deficiencies affect the body in the same way. The relation of nutrition to infection is not one problem but many, and considerable more work of a well-controlled nature is necessary before a fully adequate answer can be given to this relation. However, there are certain points which seem reasonably clear, and several results which should be mentioned.

The relation of diet to infection is not to be compared with the dramatic effects of successful vaccines, serums, or drugs; but rather with other environmental factors, such as general physical condition, sanitation, personal hygiene, and rest. There is no doubt that severe degrees of malnourishment in experimental animals, like fatigue and low body temperature, lead to an increased incidence of spontaneous infection and to increased susceptibility to certain induced infections.<sup>164</sup> This is particularly true of vitamin A deficiency, but also to some degree with other deficiencies. The high morbidity and mortality rates from infections among those with severe deficiencies indicate that this is also true for the human being.

*In Experimental Animals.* There is some evidence that mild degrees of deficiencies also lower resistance. Webster and Prichett<sup>165</sup> have found a definite difference in resistance to mouse typhoid, bichloride of mercury intoxication and botulinus toxin between two groups of mice, both of which were thought to be normal and adequately nourished as shown by satisfactory growth and procreative powers. Wooley and Sebrell<sup>166</sup> have shown that mice kept on diets inadequate in riboflavin or thiamin are more susceptible to fatal infections with pneumococcus type I inoculated by the intranasal route than were mice fed on diets containing enough of these vitamins for good growth. King and Menten,<sup>167</sup> and King and Sigal<sup>168</sup> have shown that guinea pigs receiving inadequate amounts of ascorbic acid succumb to smaller doses of diphtheria toxin than those receiving liberal amounts of ascorbic acid. Rinehart, *et al.*<sup>169</sup> have found that the heart lesions due to  $\beta$ -streptococcus infection in guinea pigs are greatly intensified by superimposed chronic scurvy. A lowered tissue resistance to infection has been observed in several of the deficiencies;<sup>170-172</sup> for example, in the skin lesions of riboflavin deficiency, the mouth lesions of black tongue and pellagra, and in the lesions of the eye in vitamin A deficiency.

Badger and Masunaga<sup>173</sup> report a lowered resistance to rat leprosy in thiamin deficiency. Sabin and Duffy<sup>174</sup> have shown that the constitutional barrier to the entrance (muscular route) of the neurotropic virus (vesicular stomatitis virus) which normally develops in young mice by the sixth week is delayed by the malnourishment. This effect was shown by general undernourishment, i.e., low calories; also by B-complex and vitamin E deficiency. However, once the barrier had developed, neither thiamin, riboflavin, nor vitamin E deficiency had an effect. Riboflavin deficiency,<sup>175</sup> even in a relatively early state, greatly lowers the resistance of rats to endemic typhus, thereby resulting in a fatal disease. Animals severely deficient in vitamin A are as resistant as normal animals. No other virus or rickettsial disease has as yet been found to react this way, so this seems to be an example of a specific and extremely severe lowering of resistance. In general, malnourishment has shown quite the opposite effect in relation to virus infections.<sup>164</sup> Recently Foster, *et al.*<sup>176</sup> and Rasmussen, *et al.*<sup>177</sup> have reported that well nourished mice are more susceptible to murine poliomyelitis than those deficient in thiamin or calories. Perhaps this is another confirmation of the close symbiotic relationship of the viruses to man.

Trager<sup>178</sup> reports a decreased resistance of chickens and ducks to *Plasmo-*



*dium lophurae* as a result of mild biotin deficiency. Some degree of specificity of biotin is shown by the fact that general debility due to other deficiencies does not give the effect.

From numerous animal experiments<sup>179-184</sup> it seems well established that diet is an important factor affecting the reaction of the host to the establishment and maintenance of hookworm and other such parasitic infestations. It has been shown in dogs and rats that injury to the host by the worm may not only be reduced (i.e., anemia) but that in many cases the infestation may be eliminated by improvement in diet alone. This supports the repeated observations and contentions<sup>184-185</sup> of many that poor nutrition is a very important conditioning factor in the epidemiology of hookworm and related parasites in man.

*In Man.* Such direct evidence on the relation of mild deficiency states to resistance to infection in man is lacking, but there are several bits of evidence which should be considered. The incidence and duration of illness in a low-income group as measured by loss of time from work and hospital days have been reported as being significantly higher in a low-income than in a higher-income group.<sup>186</sup> Harper, *et al.*<sup>187</sup> have reported a statistically significant decrease in loss of time due to minor illnesses of a group of 70 R.A.F. cadets as a result of supplementing their regular diet with vitamins A, C, and D. The prevalence of infectious diseases among the poorly-nourished populations of the world is striking and cannot be explained by sanitation, personal hygiene, and insect carriers.<sup>164</sup> Orr and Gilks<sup>124</sup> in the studies previously mentioned found that 37 percent of the tribe living on a poor diet suffered from chronic respiratory infections; while in the better-nourished tribe living under the same climatic conditions, this type of illness was found in only about 4 percent. Tuberculosis, tropical ulcers and intestinal parasites were also low in the better-nourished group. The tribe habitually eating a poor diet showed a marked drop in morbidity and mortality rate within a year after their dietary had been improved. Like observations have been reported from India and other parts of the world.

Without questioning the presence of infection and climate as factors in rheumatic fever, there is considerable evidence that an additional factor such as poor nutrition, found to be more prevalent in low-income groups, is involved.<sup>164, 169, 188</sup> The importance of good nutrition to resistance and recuperative power is recognized by most physicians and surgeons who give attention to this point, as well as rest, as part of general treatment. Also, in their experience, all other factors being equal, the poorly-nourished patients constitute the greatest risks.

#### APPLICATION OF THE SIGNIFICANCE OF OPTIMUM NUTRITION

In considering the application of existing knowledge on the significance of optimum nutrition for health and well-being, it is pertinent to mention that it is difficult to sell the idea prophylaxis, especially if its benefits lack drama, or if they appear slowly over a long period of time. Then too, the early evidence for the importance of a biological science to preventive medicine is usually founded

on animal experiments. Later, statistical measurements may be necessary to show conclusively the benefits to man. Also, such human studies require such a large program that progress is slow. To the average mind neither animal experiment nor statistics has a very strong emotional or personal appeal, and slow acceptance of this type of evidence is to be expected. But, in addition, there is a mistake which appears to be more or less prevalent and which indicates an insufficient understanding of this type of evidence but is serious because it has, without a doubt, retarded the acceptance of the newer knowledge of nutrition in some quarters. This is the mistake of supposing that an unsound procedure is involved in carrying over the findings from one species to another. Whereas, the fact is that the application of animal experiments to human problems in nutrition depends upon the same assumptions that have been used with such success in the developments of physiology and bacteriology, and which are the foundation on which the practice of medicine is built.

On the other hand, some persons may expect a good diet or improvement of nutritional status by therapy to confer absolute protection against all infectious and metabolic diseases. In any relationship between nutritional status and infection, it is evident that nutrition is only one consideration, that such factors as exposure and virulence are not to be disregarded. Hence, whatever protection is conferred by favorable nutritional status may be expected to be in terms of probability rather than certainty.<sup>189</sup>

There is no denying that there have been some premature or exaggerated claims for the benefits of optimum as compared with just adequate nutrition, that the literature contains reports of many poorly-conducted experiments and interpolations, and that there are those who make a living by exploitation. But these human failings should not produce a reaction which retards the acceptance of the real merits of the science. Much more knowledge of the relation of nutrition to health is desirable and is being uncovered at a rapid rate. Nevertheless, keeping in mind the view that buoyant health, not just passable health, is the objective of modern health philosophy and practice, the evidence now available, incomplete as it may seem, leads to but one conclusion. It is that there is a real difference, as measured in terms of growth, development, and general health record between optimum and just adequate nutrition; and that every practical effort should be made to apply this knowledge in the interest of human welfare.



## SUMMARY AND CONCLUSIONS

All the evidence from numerous surveys over the past ten years to the present among persons of all ages in many localities is without exception in complete agreement that inadequate diets are widespread in the nation. Although an appreciable percentage of the diets failing to meet the Council's recommended dietary allowances were more than 50 percent deficient in amounts of the several essential nutrients, most of the diets were less than 50 percent deficient. Accordingly, there is widespread prevalence of moderately deficient diets.

It has long been recognized that other circumstances besides deficient diet impair nutrition. Certain so-called conditioning factors, such as physical exertion, light, trauma of several kinds, and disease, raise the requirements for essentials. Therefore, adequate intake is an individual matter related to the conditioning factors in operation. If these factors are sufficiently severe, even optimum intake may not completely protect against their force during the period of maximum operation. It is evident that many factors affect nutritional status.

But evidence that deficient diet or conditioning factors are in operation at some particular point of time gives only a slight and incomplete conception of the full scope of their influence and effects. The frequency and duration of their operation is extremely important. Thus time, which for persons is age, is a significant factor. Few people eat an adequate diet every day during their lifetime. Rather, there are numerous or protracted periods when the diet is faulty. Similarly, few people escape the operation of conditioning factors at frequent or lengthy periods in their life. The repeated or protracted operation of faulty diet or various conditioning factors slowly leads to cumulative effects in deficiency states.

All the data from numerous surveys with new methods among persons of all ages in many regions are entirely in accord in showing that deficiency states are rife throughout the nation. Relatively few are the traditional severe, acute types. Most are milder in intensity and gradual in their course. Predominantly they are subacute or chronic states: some marked, but very many mild or moderate.

From this evidence it is clear that there is both a preventive and corrective problem. On the preventive side, it is evident that production of sufficient food should be maintained and that more effective distribution of proper food is needed. For the latter, it would seem advisable to give further consideration to the program of judicious enrichment of appropriate foods since that would add much to the guarantee of successful nutrition. It is also evident that diet education must be intensified and extended to the utmost, and raised to new heights of effectiveness.

On the corrective side, there is need for detection and therapeutic treatment of deficiency states among the population. For this project it is necessary to

disseminate the new diagnostic methods among the medical and public health professions. Foremost among the steps in this direction would be: (1) preparation of a handbook on methods of detecting deficiency states; (2) establishment of training centers for instruction in the medical aspects of nutrition, especially diagnosis of the deficiency states; and (3) introduction of adequate courses on nutrition, particularly its clinical aspects, into medical schools.



## REFERENCES

1. Wiehl, Dorothy, G.: Diets of Low-Income Families in New York City. *Quarterly Bulletin of the Milbank Memorial Fund*, 11: 308-324 (No. 4, Oct.), 1933.
2. Wiehl, Dorothy G.: Diets of Low-Income Families Surveyed in 1933. *Health and Depression Studies No. 3. Public Health Reports*, 51: 77-97 (No. 4, Jan. 24), 1936.
3. Wiehl, Dorothy G.: Diets of Urban Families with Low Incomes. An Analysis of Weekly Food Budgets in 472 Families in Baltimore, Cleveland, Detroit, Pittsburgh, and Syracuse in April-May 1933. *Milbank Memorial Fund Quarterly*, 12: 343-369 (No. 4, Oct.), 1934.
4. Stiebeling, Hazel K. and Phipard, Esther F.: Diets of Families of Employed Wage Earners and Clerical Workers in Cities. U. S. Department of Agriculture, Circular No. 507, Washington, Jan. 1939.
5. Stiebeling, Hazel K.; Monroe, Day; Coons, Callie M.; Phipard, Esther F.; and Clark, Faith: Family Food Consumption and Dietary Levels, Five Regions. *Consumer Purchases Study, Farm Series*. U. S. Department of Agriculture, Misc. Publication No. 405, Washington, 1941.
6. Stiebeling, Hazel K.; Monroe, Day; Phipard, Esther F.; Adelson, Sadye F.; and Clark, Faith: Family Food Consumption and Dietary Levels, Five Regions. *Consumer Purchases Study, Urban and Village Series*. U. S. Department of Agriculture, Misc. Publication No. 452, Washington, 1941.
7. Recommended Dietary Allowances. National Research Council, Reprint and Circular Series No. 115, Washington, D. C., Jan. 1943.
8. Food and Drugs. *Federal Register*, 6: 5922, 5925 (No. 227, Nov. 22), 1941, Title 21.
9. Stiebeling, Hazel K. and associates: Unpublished data.
10. Stiebeling, Hazel K.: Food Consumption Studies and Dietary Recommendations. *Federation Proceedings*, 1: 327-330 (No. 3, Sept.), 1942.
11. Britton, Virginia: Food Consumption of 538 Farm and 299 Village Families in Vermont. Vermont Agricultural Experiment Station, Bulletin No. 474, Burlington, Vt., June 1941.
12. Sanders, Agnes B. and Mack, Pauline B.: Unpublished data.
13. Mack, Pauline B. and associates: Unpublished data.
14. Wiehl, Dorothy G. and Palmer, C. E.: Summer Diets of the Poor in Washington, D. C. *Milbank Memorial Fund Quarterly*, 17: 5-28 (No. 1, Jan.), 1939.
15. Murphy, Elizabeth: A Study of Vitamin C Nutrition in a Group of School Children. II. Dietary Evaluation. *Journal of Nutrition*, 21: 527-539 (No. 5, May 10), 1941.
16. Reynolds, May S.; Ohlson, Margaret A.; Pittman, Martha S.; McKay, Hughina; Patton, Mary Brown; Donelson, Eva; Leverton, Ruth; Meiller, Ella J.; and Bitting, Mary H.: The Dietary Habits of College Students. *Journal of Home Economics*, 34: 379-384 (No. 6, June), 1942.
17. Smith, Janice M. and associates: Unpublished data.
18. Wiehl, Dorothy G.: Medical Evaluation of Nutritional Status. VII. Diets of High School Students of Low Income Families in New York City. *Milbank Memorial Fund Quarterly*, 20: 61-82 (No. 1, Jan.), 1942.
19. Wiehl, Dorothy G.: Unpublished data.
20. Grigsby, Nora E.; McBryde, Laureame C.; and Davis, H. J.: A Study of the Adequacy of Diets of Farm Security Administration Families in Louisiana. Nutrition Laboratories, College of Agriculture, Louisiana State University, Baton Rouge, Louisiana. Mimeographed Leaflet, Sept. 15, 1942.
21. Kelly, H. T. and Sheppard, Myrtle: A Dietary Study of Subjects from Upper Income Groups. *New England Journal of Medicine*, 228: 118-124 (Jan. 28), 1943.

22. Hardy, Martha C.; Spohn, Adelaide; Austin, Gertrude; McGiffert, Sarah; Mohr, Edna; and Peterson, Agnes B.: Nutritional and Dietary Inadequacies Among City Children from Different Socio-Economic Groups. *Journal of the American Dietetic Association*, **19**: 173-181 (No. 3, March), 1943.
23. Milam, D. F.: A Nutrition Survey of a Small North Carolina Community. *American Journal of Public Health*, **32**: 406-412 (April), 1942.
24. Milam, D. F.: Unpublished data.
25. Youmans, John B.; Patton, E. White; and Kern, Ruth: Surveys of the Nutrition of Populations. *American Journal of Public Health*, **32**: 1371-1379 (No. 12, Dec.), 1942.
26. Youmans, John B.; Patton, E. White; and Kern, Ruth: Surveys of the Nutrition of Populations. *American Journal of Public Health* **33**: 58-72 (No. 1, Jan.), 1943.
27. Youmans, John B.; Patton, E. White; Kern, Ruth; Sutton, W. R.; and Steinkamp, Ruth: Unpublished data.
28. Clayton, Mary M.: A Study of the Food Habits and Nutritional Status of Children in Selected Communities in Maine. *The Maine Agricultural Experiment Station Bulletin No. 405*, pp. 431-437, Orono, Me., June 1941.
29. Clayton, Mary M.: The Nutritional Status of University of Maine Freshman Girls as Related to Their Diets. *Maine Agricultural Experiment Station Bulletin No. 405*, pp. 437-438, Orono, Me., June 1941.
30. Willard, W. R.: Working for Better Nutrition in a Rural Community. *American Journal of Public Health*, **32**: 996-1000 (Sept.), 1942.
31. Bryson, Bertha E.; Tucker, Clara; and Davis, H. J.: A Study of the Diets of Home Demonstration Club Members' Families in Twenty-Seven Parishes of Louisiana. *Louisiana Bulletin No. 356*, Louisiana State University Agricultural Experiment Station, Baton Rouge, La., Dec. 1942.
32. Coco, Lucille D.; Moore, Margaret; Goldsmith, Grace A.; Lucas, G. P.; and Davis, H. J.: A Study of the Adequacy of Diets Consumed by Grade-School and High-School Students in Louisiana. *Louisiana Bulletin No. 360*, Louisiana State University Agricultural Experiment Station, Baton Rouge, La., Jan. 1943.
33. Downes, Jean: A Study of Food Habits of Tuberculous Families in a Harlem Area of New York City. *Milbank Memorial Fund Quarterly*, **21**: 164-181 (No. 2, April), 1943.
34. Wiehl, Dorothy G.: Diets of a Group of Aircraft Workers in Southern California. *Milbank Memorial Fund Quarterly*, **20**: 329-336 (No. 4, Oct.), 1942.
35. Mack, Pauline B.; Urbach, Charles; Smith, Janice M.; Logan, Catherine H.; Rose, Elizabeth K.; Stewart, A. H.; and Dodds, Paul: A Contribution to the Study of Nutritional Status in Rural and Urban Populations. *Pennsylvania State College Bulletin*, **36**: (No. 52), 1942.
36. Gallup, George: Report from American Institute of Public Opinion. *New York Times*, Feb. 8, 1943.
37. Stiebeling, Hazel K.: Adequacy of American Diets. *Journal of the American Medical Association*, **121**: 831-838 (No. 11, March 13), 1943.
38. Sherman, H. C.: *Chemistry of Food and Nutrition*. Sixth Edition. New York, Macmillan Company, 1941, p. 381.
39. Bing, F. C.: Foods as Sources of the Vitamins. *Federation Proceedings*, **1**: 296-303 (No. 3, Sept.), 1942.
40. Lanford, Caroline S. and Sherman, H. C.: Nutrition, 1941 and 1942. *Annual Review of Biochemistry*, **12**: 397-424, Stanford University, California, 1943.
41. Kruse, H. D.: A Concept of the Deficiency States. *Milbank Memorial Fund Quarterly*, **20**: 245-261 (No. 3, July), 1942.
42. Jolliffe, Norman: Conditioned Malnutrition. *Journal of the American Medical Association*, **122**: 299-306 (No. 5, May 29), 1943.
43. Kruse, H. D.: Medical Evaluation of Nutritional Status. *Journal of the American Medical Association*, **121**: 584-591 (No. 8, Feb. 20), 1943; and **121**: 669-677 (No. 9, Feb. 27), 1943.



44. Jolliffe, N.; McLester, J. S.; and Sherman, H. C.: The Prevalence of Malnutrition. *Journal of the American Medical Association*, **118**:944-950 (No. 12, March 21), 1942.
45. Lowe, R. C.: Vitamin Deficiency States in Louisiana. *New Orleans Medical & Surgical Journal*, **95**:407-414 (No. 9, March), 1943.
46. Goldsmith, Grace A.: The Incidence and Recognition of Riboflavin and Niacin Deficiency in Medical Diseases. *Southern Medical Journal*, **36**:108-116 (No. 2, Feb.), 1943.
47. Krupp, Marcus A.: The Incidence of Nutritional and Vitamin Deficiency. *Journal of the American Medical Association*, **119**:1475-1479 (No. 18, Aug. 29), 1942.
48. Bethell, F. H.; Gardiner, S. H.; and MacKinnon, F.: The Influence of Iron and Diet on the Blood in Pregnancy. *Annals of Internal Medicine*, **13**:91-100 (July), 1939.
49. Labate, J. S.: Classification and Treatment of the Anemias of Pregnancy. *American Journal of Obstetrics and Gynecology*, **38**:48-56 (July), 1939.
50. Eskridge, J. B. and Serwer, M. J.: Blood Studies in Private Obstetrical Cases. *Southern Medical Journal*, **32**:24-30 (Jan.), 1939.
51. Gordon, H.: Anemia as a Public Health Problem. *Kentucky Medical Journal*, **38**:415-417 (Sept.), 1940.
52. Wiehl, Dorothy G. and Kruse, H. D.: Medical Evaluation of Nutritional Status. V. Prevalence of Deficiency Diseases in Their Subclinical Stages. *Milbank Memorial Fund Quarterly*, **19**:241-251 (No. 3, July), 1941.
53. Kruse, H. D.: Personal communication.
54. Zayaz, S. L.; Mack, P. B.; Sprague, P. K.; and Bauman, A. W.: Nutritional Status of School Children in a Small Industrial City. *Child Development*, **11**:1-25 (Jan.), 1940.
55. Mack, Pauline B.; Smith, Janice M.; Logan, Catherine H.; O'Brien, Anne T.; Shaw, John J.; and Dodds, Paul: Hemoglobin Values in Pennsylvania Mass Studies in Human Nutrition. *Milbank Memorial Fund Quarterly*, **19**:282-303 (No. 3, July), 1941.
56. Scott, J. R. and Janeway, M. M.: Nutritional Study: Analysis of 100 Medical Admissions to St. Luke's Hospital, New York City. *New York State Journal of Medicine*, **40**:440-443 (March 15), 1940.
57. Abbott, O. D. and Ahmann, C. F.: Iron Deficiency in Anemia in Children. *American Journal of Diseases of Children*, **58**:811-816 (Oct.), 1939.
58. Youmans, John B.; Patton, E. White; and Kern, Ruth: Surveys of the Nutrition of Populations. Unpublished data.
59. Borsook, Henry; Alpert, Elmer; and Keighley, Geoffrey L.: Nutritional Status of Aircraft Workers in Southern California. *Milbank Memorial Fund Quarterly*, **21**:115-157 (No. 2, April), 1943.
60. Harris, R. S.; Weeks, E.; and Kinde, M.: Effect of a Supplementary Food on the Nutritional Status of School Children. *Journal of the American Dietetic Association*, **19**:182-189 (March), 1943.
61. Williams, R. D. and Wilder, R. M.: The Prevalence of Malnutrition in the American Population: A Review of the Evidence. *National Research Council Report*, 1941.
62. Crane, Marian M. and Woods, Philip W.: A Study of Vitamin C Nutrition in a Group of School Children. *New England Journal of Medicine*, **224**:503-509 (No. 12, March 20), 1941.
63. Minot, A. S.; Dodd, K.; Keller, M.; and Frank, H.: A Survey of the State of Nutrition with Respect to Vitamin C in a Southern Pediatric Clinic. *Journal of Pediatrics*, **16**:717-728 (June), 1940.
64. Holmes, F. E.; Cullen, G. E.; and Nelson, W. E.: Levels of Ascorbic Acid in Blood Plasma of Apparently Healthy Children. *Journal of Pediatrics*, **18**:300-309 (March), 1941.
65. Fincke, M. L.: The Ascorbic Acid (Vitamin C) Metabolism of College Students. A Cooperative Study. *Journal of the American Dietetic Association*, **16**:325-328 (April), 1940.

66. Milam, D. F. and Wilkins, Walter: Plasma Vitamin C Levels in a Group of Children Before and After Dietetic Adjustment. *American Journal of Tropical Medicine*, **21**: 487-491 (May), 1941.
67. Holman, E. F.: Vitamin and Protein Factors in Pre-operative and Post-operative Care of Surgical Patients. *Surgery, Gynecology and Obstetrics*, **70**: 261-269 (Feb. 15), 1940.
68. Croft, J. D. and Snorf, L. D.: Cevitamic Acid Deficiency. *American Journal of the Medical Sciences*, **198**: 403-408 (Sept.), 1939.
69. Jolliffe, Norman: *Preventive Medicine in Modern Practice*. New York, Paul Hoeber, Chapter 5, pp. 79-122, 1942.
70. Schnedorf, J. D.; Weber, C. J.; and Clendening, L.: Vitamin Survey of Normal Industrial Workmen. *American Journal of Digestive Diseases*, **9**: 188-191 (June), 1942.
71. Manning, I. H., Jr. and Milam, D. F.: Medical and Nutritional Survey of 800 N.Y.A. Youths; Experience at National Youth Administration Work Center in Physical Rehabilitation of Youths of Draft Age. *Southern Medical Journal*, **36**: 373-380 (No. 5, May), 1943.
72. Feder, Virginia H.; Lewis, G. T.; and Alden, H. S.: Urinary Riboflavin. Unpublished data.
73. Kruse, H. D.; Sydenstricker, V. P.; Sebrell, W. H.; and Cleckley, H. M.: Ocular Manifestations of Ariboflavinosis. *Public Health Reports*, **55**: 157-169 (No. 4, Jan. 26), 1940.
74. Sydenstricker, V. P.; Sebrell, W. H.; Cleckley, H. M.; and Kruse, H. D.: The Ocular Manifestations of Ariboflavinosis. *Journal of the American Medical Association*, **114**: 2437-2445 (June 22), 1940.
75. Johnson, L. V. and Eckardt, R. E.: Rosacea Keratitis and Conditions with Vascularization of the Cornea Treated with Riboflavin. *Archives of Ophthalmology*, **23**: 899-907 (May), 1940.
76. Mack, Pauline B.; Logan, Catherine H.; and associates: Unpublished data.
77. Sebrell, W. H.: Vitamins and Public Health. *Federation Proceedings*, **1**: 319-323 (No. 3, Sept.), 1942.
78. Jolliffe, Norman and Goodhart, Robert: Vitamins in the Practice of Medicine. *Federation Proceedings*, **1**: 316-319 (No. 3, Sept.), 1942.
79. Youmans, J. B.; Patton, E. W.; Robinson, W. D.; and Kern, Ruth: An Analysis of Corneal Vascularity as Found in a Survey of Nutrition. *Transactions of the Association of American Physicians*, **57**: 49-54, 1942.
80. Sandstead, H. R.: Superficial Vascularization of the Cornea. The Results of Riboflavin Therapy. *Public Health Reports*, **57**: 1821-1825 (Nov. 27), 1942.
81. Tisdall, F. F.; McCreary, J. F.; and Pearce, H.: The Effect of Riboflavin on Corneal Vascularization and Symptoms of Eye Fatigue in R.C.A.F. Personnel. *Canadian Medical Association Journal*, **49**: 5-13 (July), 1943.
82. Youmans, J. B. and Patton, E. W.: The Laboratory Diagnosis of Nutritional Deficiencies. *Clinics*, **1**: 303-345 (Aug.), 1942.
83. Sydenstricker, V. P.; Kelly, A. R.; and Weaver, J. W.: Ariboflavinosis, with Special Reference to the Ocular Manifestations. *Southern Medical Journal*, **34**: 165-170 (Feb.), 1941.
84. Kruse, H. D. and Tisdall, F. F.: Proceedings of the Twentieth Annual Conference of the Milbank Memorial Fund, pp. 35 and 36, May 1942.
85. Mori, S.: Primary Changes in Eyes of Rats Which Result from Deficiency of Fat-Soluble A in Diet. *Journal of the American Medical Association*, **79**: 197-200 (July 15), 1922.
86. Bessey, O. A. and Wolbach, S. B.: Vitamin A, Physiology and Pathology. *Journal of the American Medical Association*, **110**: 2072-2080 (June 18), 1938.
87. Kruse, H. D.: Medical Evaluation of Nutritional Status. IV. The Ocular Manifestations of Avitaminosis A, with Especial Consideration of the Detection of Early Changes by Biomicroscopy. *Milbank Memorial Fund Quarterly*, **19**: 207-240



- (No. 3, July), 1941; and Public Health Reports, 56: 1301-1324 (No. 26, June 27), 1941.
88. Berliner, M. L.: Regarding the Early Detection of Avitaminosis A by Gross or Biomicroscopic Examination of the Conjunctiva. *American Journal of Ophthalmology*, 25: 302-308 (No. 3, March), 1942.
  89. Van Veen, A. G.: The Vitamin Level of the Organism and its Determination. *Mededeel v. d. dienst d. Volksgezondh. in Nederl.-Indië*, 26: 300-307, 1937.
  90. Patek, A. J., Jr. and Haig, Charles: Effect of Administration of Thyroid Extract and of Alpha-Dinitrophenol upon Dark Adaptation. *Proceedings of the Society for Experimental Biology and Medicine*, 46: 180-182 (Jan.), 1941.
  91. Kruse, H. D.: Detection of Avitaminosis A by Gross and Biomicroscopic Examination of the Conjunctiva. *American Journal of Ophthalmology*, 25: 1386-1391 (No. 11, Nov.), 1942.
  92. Boyd, William: Quoted by Piersol, G. M.: Arteriosclerosis: Social Significance and Recent Advances in Treatment. *Bulletin of The New York Academy of Medicine*, 18: 36-52 (Jan.), 1942.
  93. Jolliffe, Norman and Stern, Marvin: Objective Manifestations of Nutritional Deficiency Diseases. *Clinics*, 1: 282-302 (Aug.), 1942.
  - 93a. Getz, H. R.: Unpublished data.
  94. Hess, A. F.: *Rickets Including Osteomalacia and Tetany*. Philadelphia, Lea & Febiger, 485 pp., 1929.
  95. Youmans, J. B.: *Nutritional Deficiencies—Diagnosis and Treatment*. J. B. Lippincott Company, 398 pp., 1941.
  96. Levy, Julius and Silver, H. B.: Can Rickets be Eliminated from a Large City? *Archives of Pediatrics*, 56: 96-105 (Feb.), 1939.
  97. Rhoads, Teresa F.; Rapoport, Milton; Kennedy, Ruth; and Stokes, Joseph, Jr.: Studies on Growth and Development of Male Children Receiving Evaporated Milk; Effects of Various Vitamin Supplements on Growth and the Incidence of Rickets. *Journal of Pediatrics*, 19: 169-189 (Aug.), 1941.
  98. Follis, R. H.; Jackson, Deborah A.; and Park, E. A.: The Problem of the Association of Rickets and Scurvy. *American Journal of the Diseases of Children*, 60: 745-747, 1940.
  99. Kruse, H. D.: The Lingual Manifestations of Aniacinosis, with Especial Consideration of the Detection of Early Changes by Biomicroscopy. *Milbank Memorial Fund Quarterly*, 20: 262-289 (No. 3, July), 1942.
  100. Kruse, H. D.: The Gingival Manifestations of Avitaminosis C, with Especial Consideration of the Detection of Early Changes by Biomicroscopy. *Milbank Memorial Fund Quarterly*, 20: 290-323 (No. 3, July), 1942.
  101. Eddy, W. H. and Dalldorf, G.: *The Avitaminoses*. Baltimore, Williams and Wilkins Company, 473 pp., 1941.
  102. Wolbach, S. B. and Bessey, O. A.: Tissue Changes in Vitamin Deficiencies. *Physiological Reviews*, 22: 233-289 (July), 1942.
  103. Sebrell, W. H.: The Nature of Nutritional Diseases Occurring in the South. *Milbank Memorial Fund Quarterly*, 17: 358-366 (No. 4, Oct.), 1939.
  104. Field, Henry, Jr.; Parnall, C., Jr.; and Robinson, W. D.: Pellagra in the Average Population of the Northern States. *New England Journal of Medicine*, 223: 307-315 (Aug. 29), 1940.
  105. Abercrombie, T. F.: Public Health Problems and Their Relation to Medical Care in Georgia. *Journal of the Medical Association of Georgia*, 29: 343-346 (July), 1940.
  106. Clendening, Logan: The National Nutrition. *Journal of the American Medical Association*, 117: 1035-1036 (No. 12, Sept. 20), 1941.
  107. Minot, G. R.: Nutritional Deficiency. *Annals of Internal Medicine*, 12: 429-442, 1938.
  108. Stieglitz, E. J.: The Rôle of Health Education in the Promotion of Optimal Health and in the Retardation of Degenerative Diseases. A Venture in Public Health Integration. The 1941 Health Education Conference of the New York Academy of Medicine. *Columbia University Press*, 1942.

109. Williamson, G. S. and Pearse, I. H.: Biologists in Search of Material—An Interim Report on the Work of the Pioneer Health Centre, Peckham. Faber and Faber, London, pp. 9-104, 1938.
110. Editorial: We Have Come a Long Way in Nutrition. *American Journal of Public Health*, **31**: 630-631 (No. 6, June), 1941.
111. Boudreau, Frank G.: International and National Aspects of the Campaign for Better Nutrition. *Journal of the American Dietetic Association*, **15**: 885-893 (No. 10, Dec.), 1939.
112. Orr, J. B.: Nutrition and Human Welfare. *Nutrition Abstracts and Reviews*, **11**: 3-11 (July), 1941.
113. Sherman, H. C. and Campbell, H. L.: Nutritional Well-Being and Length of Life as Influenced by Different Enrichments of an Already Adequate Diet. *Journal of Nutrition*, **14**: 609-620, 1937.
114. Sherman, H. C.; Campbell, H. L.; and Lanford, C. S.: Experiments on the Relation of Nutrition to the Composition of the Body and the Length of Life. *Proceedings of the National Academy of Science*, **25**: 16-20 (Jan.), 1939.
115. Sherman, H. C. and Ellis, L. N.: Necessary Versus Optimal Intake of Vitamin G (B<sub>2</sub>). *Journal of Biological Chemistry*, **104**: 91-97 (Jan.), 1934.
116. Ellis, L. N.; Zmachinsky, Anna; and Sherman, H. C.: Experiments upon the Significance of Liberal Levels of Intake of Riboflavin. *Journal of Nutrition*, **25**: 153-160 (Feb.), 1943.
117. Sherman, H. C.: *Chemistry of Food and Nutrition*. Sixth Edition. New York, Macmillan Company, 1941, 611 pp.
118. Sherman, H. C.: *The Science of Nutrition*. Columbia University Press, New York, 1943, 253 pp.
119. Waterman, R. E. and Ammerman, M.: Studies of Crystalline Vitamin B; 5. The Effect of Graduated Doses on Growing Rats. *Journal of Nutrition*, **10**: 35-44 (July), 1935.
120. U. S. Department of Agriculture: Food and Life. Yearbook of Agriculture. U. S. Government Printing Office, House Document No. 28, 1165 pp., 1939.
121. McCarrison, R.: Good Diet and Bad One; Experimental Contrast. *Indian Journal of Medical Research*, **14**: 649-654 (Jan.), 1927.
122. McCarrison, R.: Problems of Nutrition in India. *Nutrition Abstracts and Reviews*, **2**: 1-8 (No. 1, July), 1932.
123. Radhakrishna Rao, M. V.: Intestinal Changes in Monkeys Fed on Poor Rice Diets. *Indian Journal of Medical Research*, **30**: 273-284 (April), 1942.
124. Orr, J. B. and Gilks, J. L.: Studies of Nutrition. The Physique and Health of Two African Tribes. Medical Research Council. Special Report Series No. 155, 82 pp., 1931.
125. Mann, H. C. C.: Diets for Boys During the School Age. Medical Research Council. Special Report Series No. 105, 81 pp., 1926.
126. Orr, J. B.: Milk Consumption and the Growth of School-Children. *Lancet*, **I**: 202-203 (Jan. 28) 1928.
127. Leighton, G. and McKinlay, P. L.: Milk Consumption and the Growth of School Children. Report on an Investigation in Lanarkshire Schools. Edinburgh, Department of Health for Scotland. His Majesty's Stationery Office, 20 pp., 1930.
128. Borland, V.: School Meals and the Oslo Breakfast. *Journal of the Royal Institute of Public Health and Hygiene*, **3**: 149-156 (June), 1940.
129. The School Lunch. *Hygeia*, **19**: 669-670 (Aug.), 1941.
130. Rowntree, L. G.; McGill, K. H.; Folk, O. H.: Health of Selective Service Registrants. *Journal of the American Medical Association*, **118**: 1223-1227 (April 4), 1942.
131. Crawford, J. A.: The Work at the Recruits' Physical Development Depot, Canterbury. "The Undersized Recruit." *Journal of the Royal Army Medical Corps*, **73**: 1-39 (July), 1939.
132. A Height and Weight Survey of Toronto Elementary School Children. Department of Trade and Commerce, Dominion Bureau of Statistics, Social Analysis Branch, Ottawa, Canada, 1939.



133. Boynton, B.: The Physical Growth of Girls: A Study of the Rhythm of Physical Growth from Anthropometric Measurements on Girls Between Birth and Eighteen Years. University of Iowa Studies in Child Welfare, **12**: no. 4, 105 pp. ment Abstracts, **11**: 2, 1936.
134. Paton, D. N. and Findlay, L.: Poverty, Nutrition and Growth: Studies of Child Life in Cities and Rural Districts of Scotland. Medical Research Council. Special Report Series No. 101, 333 pp., 1926.
135. Ebbs, J. H.; Tisdall, F. F.; and Scott, W. A.: The Influence of Prenatal Diet on the Mother and Child. Journal of Nutrition, **22**: 515-526 (No. 5, Nov.), 1941.
136. Peoples League of Health; Nutrition of Expectant and Nursing Mothers. Lancet, **II**: 10-12 (July 4), 1942.
137. Williams, R. D.; Mason, H. L.; Smith, B. F.; and Wilder, R. M.: Induced Thiamine (vitamin B<sub>1</sub>) Deficiency and the Thiamine Requirement of Man; Further Observations. Archives of Internal Medicine, **69**: 721-738 (May), 1942.
138. Jeghers, H.: Medical Progress; Nutrition; Appearance of the Tongue as an Index of Nutritional Deficiency. New England Journal of Medicine, **227**: 221-228 (Aug. 6), 1942.
139. Rosenblum, L. A. and Jolliffe, N.: The Oral Manifestations of Vitamin Deficiencies. Journal of the American Medical Association, **117**: 2245-2248 (Dec. 27), 1941.
140. Sydenstricker, V. P.: Clinical Manifestations of Ariboflavinosis. American Journal of Public Health, **31**: 344-350 (April), 1941.
141. Keys, Ancel and Henschel, A. F.: Vitamin Supplementation of U. S. Army Rations in Relation to Fatigue and the Ability to Do Muscular Work. Journal of Nutrition, **23**: 259-269 (March), 1942.
142. Foltz, E. E.; Ivy, A. C.; and Barborka, C. J.: Influence of Components of the Vitamin B-Complex on Recovery from Fatigue. Journal of Laboratory and Clinical Medicine, **27**: 1396-1399 (Aug.), 1942.
143. Simonson, E.; Enzer, N.; Baer, A.; and Braun, R.: The Influence of Vitamin B (Complex) Surplus on the Capacity for Muscular and Mental Work. Journal of Industrial Hygiene and Toxicology, **24**: 83-90 (April), 1942.
144. Egaña, E.; Johnson, R. E.; Bloomfield, R.; Brouha, L.; Meiklejohn, A. P.; Whittenberger, J.; Darling, R. C.; Heath, C.; Graybiel, A.; and Consolazio, F.: The Effects of a Diet Deficient in the Vitamin B Complex on Sedentary Men. American Journal of Physiology, **137**: 731-741 (Nov.), 1942.
145. Johnson, R. E.; Darling, R. C.; Forbes, W. H.; Brouha, L.; Egaña, E.; and Graybiel, A.: The Effects of a Diet Deficient in Part of the Vitamin B Complex Upon Men Doing Manual Labor. Journal of Nutrition, **24**: 585-596 (Dec.), 1942.
146. Barborka, C. J.; Foltz, E. E.; and Ivy, A. C.: Relationship Between Vitamin B-Complex Intake and Work Output in Trained Subjects. Journal of the American Medical Association, **122**: 717-720 (No. 11, July 10), 1943.
147. McCollum, E. V.; Orent-Keiles, E.; and Day, H. G.: The Newer Knowledge of Nutrition. New York, Macmillan Company, 701 pp., 1939.
148. Cowell, Stuart J.: Diet and Dental Disease. Nutrition Abstracts and Reviews, **5**: 567-575 (No. 3, Jan.), 1936.
149. Marshall, J. A.: Dental Caries. Physiological Reviews, **19**: 389-414 (July), 1939.
150. Howe, P. R.; White, R. L.; and Rabine, M.: Retardation of Dental Caries in Out-Patients of a Dental Infirmary. American Journal of Diseases of Children, **46**: 1045-1049 (Nov. pt. I), 1933.
151. Howe, P. R.; White, R. L.; and Elliott, M. D.: Influence of Nutritional Supervision on Dental Caries. Journal of the American Dental Association, **29**: 38-43 (Jan.), 1942.
152. Boyd, J. D.: Nutrition as it Affects Tooth Decay. Journal of the American Dietetic Association, **18**: 211-215 (April), 1942.
153. Boyd, J. D.; Drain, C. L.; and Nelson, M.: Dietary Control of Dental Caries. American Journal of Diseases of Children, **38**: 721-725 (Oct.), 1929.
154. The Influence of Diet on Caries in Children's Teeth (Final Report). By the Committee for the Investigation of Dental Disease. Medical Research Council. Special Report Series No. 211, 137 pp., 1936.

155. Mellanby, May: Diet and the Teeth. An Experimental Study. Part I. Dental Structure in Dogs. Medical Research Council. Special Report Series No. 140, 308 pp., 1929.
156. Mellanby, May: Diet and the Teeth. An Experimental Study. Part 3. The Effect of Diet on Dental Structure and Disease in Man. Medical Research Council. Special Report Series No. 191, 180 pp., 1934.
157. Furnas, C. C. and Furnas, S. M.: Man, Bread and Destiny. New York, Reynal and Hitchcock, 364 pp., 1937.
158. The Incidence of Dental Disease in Children. By the Committee for the Investigation of Dental Disease. Medical Research Council. Special Report Series No. 97, 48 pp., 1925.
159. Messner, C. T.; Gafafer, W. M.; Cady, F. C.; and Dean, H. T.: Dental Survey of School Children, Ages 6-14 Years, Made in 1933, 1934 in 26 States. Public Health Bulletin No. 226. Government Printing Office, Washington, 248 pp., May 1936.
160. King, J. D.: Dental Disease in the Island of Lewis. Medical Research Council. Special Report Series No. 241, 63 pp., 1940.
161. Koehne, M. and Bunting, R. W.: Studies in the Control of Dental Caries, II. Journal of Nutrition, 7: 657-678 (June), 1934.
162. Robertson, E. C.: The Vitamins and Resistance to Infection. Medicine, 13: 123-206 (May), 1934.
163. Clausen, S. W.: The Influence of Nutrition upon Resistance to Infection. Physiological Reviews, 14: 309-350 (July), 1934.
164. Perla, David and Marmorston, Jessie: Natural Resistance and Clinical Medicine. New York, Little Brown and Company, 1344 pp., 1941.
165. Webster, L. T.: The Rôle of Microbic Virulence, Dosage and Host Resistance in Determining the Spread of Bacterial Infection Among Mice. II. B. Enteritidis Infection. Journal of Experimental Medicine, 52: 931-948 (Dec.), 1930.
166. Wooley, J. G. and Sebrell, W. H.: Nutritional Deficiency and Infection; Influence of Riboflavin or Thiamin Deficiency on Fatal Experimental Pneumococcal Infection in White Mice. Public Health Reports, 57: 149-161 (Jan. 30), 1942.
167. King, C. G. and Menten, M. L.: The Influence of Vitamin C upon the Resistance to Diphtheria Toxin. Journal of Nutrition, 10: 129-155 (Aug.), 1935.
168. Sigal, A. and King, C. G.: Vitamin C and Diphtheria Toxin. Journal of Pharmacology and Experimental Therapeutics, 59: 468-473 (No. 4, April), 1937.
169. Rinehart, J. F.; Connor, C. L.; and Mettier, S. R.: Further Observations on Pathologic Similarities Between Experimental Scurvy Combined with Infection, and Rheumatic Fever. Journal of Experimental Medicine, 59: 97-114 (Jan.), 1934.
170. Riddle, J. W.; Spies, T. D.; and Hudson, N. P.: A Note on the Interrelationship of Deficiency Diseases and Resistance of Infection. Proceedings of the Society of Experimental Biology and Medicine, 45: 361-364 (Oct.), 1940.
171. Smith, D. T.; Persons, E. L.; and Harvey, H. I.: On the Identity of the Goldberger and Underhill Types of Canine Blacktongue. Secondary Fuso-Spirochetal Infection in Each. Journal of Nutrition, 14: 373-381 (Oct.), 1937.
172. Hetler, R. A.: The Development of Xerophthalmia and the Keratinization of Epithelial Tissue on Withdrawal of Vitamin A from the Diet of the Monkey, Guinea Pig, Rabbit and Adult Albino Rat. Journal of Nutrition, 8: 75-103 (July), 1934.
173. Badger, L. F.; Masunaga, E.; and Wolf, D.: Leprosy: Vitamin B<sub>1</sub> Deficiency and Rat Leprosy. Public Health Reports, 55: 1027-1041 (June 7), 1940.
174. Sabin, A. B. and Duffy, C. E.: Nutrition As a Factor in the Development of Constitutional Barriers to Involvement of the Nervous System by Certain Viruses. Science, 91: 552-554 (June 7), 1940.
175. Pinkerton, H. and Bessey, O. A.: The Loss of Resistance to Murine Typhus Infection Resulting from Riboflavin Deficiency in Rats. Science, 89: 368-370 (Apr. 21), 1939.
176. Foster, Claire; Jones, J. H.; Werner, Henle; and Dorfman, Freeda: Response to Murine Poliomyelitis Virus, Lansing Strain, of Mice on Different Levels of



- Thiamin Intake. Proceedings of the Society of Experimental Biology and Medicine, **51**: 215-216 (Nov.), 1942.
177. Rasmussen, A. F.; Waismann, H. A.; Elvehjem, C. A.; and Clark, P. F.: Rôle of Nutrition in Response of Host to Poliomyelitis Virus Infection. *Journal of Bacteriology*, **45**: 85-86 (Jan.), 1943.
178. Trager, William: The Influence of Biotin upon Susceptibility to Malaria. *Science*, **97**: 206-207 (Feb. 26), 1943.
179. Watt, J. Y. C.; Golden, W. R. C.; Olason, Fridgeir; and Mladinich, George: The Relation of Vitamin A to Resistance to *Nippostrongylus Muris*. *Science*, **97**: 381-382 (April 23), 1943.
180. Otto, G. F. and Landsberg, J. W.: Dietary Deficiencies and Iron Salts in Hookworm Infections. *American Journal of Hygiene, Sec. D*, **31**: 37-47 (March), 1940.
181. Foster, A. O. and Cort, W. W.: Further Studies on the Effect of a Generally Deficient Diet upon the Resistance of Dogs to Hookworm Infestation. *American Journal of Hygiene*, **21**: 302-318 (No. 2, Mar.), 1935.
182. Lawler, H. J.: The Relation of Vitamin A to Immunity to *Strongyloides* Infection. *American Journal of Hygiene, Sec. D*, **34**: 65-72 (Nov.), 1941.
183. McCoy, O. R.: The Effect of Vitamin A Deficiency on the Resistance of Rats to Infection with *Trichinella Spiralis*. *American Journal of Hygiene*, **20**: 169-180 (July), 1934.
184. Cort, W. W. and Otto, G. F.: Immunity in Hookworm Disease. *Review of Gastroenterology*, **7**: 2-14 (Jan.-Feb.), 1940.
185. Rhoads, C. P.; Castle, W. B.; Payne, G. C.; and Lawson, H. A.: Observations on the Etiology and Treatment of Anemia Associated with Hookworm Infection in Puerto Rico. *Medicine*, **13**: 317-375 (Sept.), 1934.
186. A National Health Program and Some Proposals Toward Design. *Journal of the American Medical Association*, **110**: 656-665 (Feb. 26), 1938.
187. Harper, A. A.; Mackay, I. F. S.; Raper, H. S.; and Camm, G. L.: Vitamins and Physical Fitness. *British Medical Journal*, 243-245 (Feb. 27), 1943.
188. Coburn, A. F. and Moore, L. V.: Nutrition as a Conditioning Factor in the Rheumatic State. *American Journal of the Diseases of Children*, **65**: 744-756 (Feb.), 1943.
189. Kruse, H. D.: Nutritional Needs of American Youth. *American Journal of Public Health*, **33**: 249-255 (No. 3, March), 1943.





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